THE SCIENTIFIC BASIS FOR THE MODELLING OF CARIES PREVENTIVE STRATEGIES

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'..the proposition that the soul is in harmony has not been demonstrated at all but rests only on probability...'

Simmias to Socrates

Phaedo

ABSTRACT

The decline in dental caries in many industrialised countries has prompted a re-assessment of preventive strategies for dental caries. Although methods to prevent dental caries are well established, few data exist on defining the most appropriate combination of preventive methods to be used for differing levels of dental caries. Texts that do outline preventive methods do not make recommendations on the dental caries conditions under which they should be used. The most popular approach is the identification of individuals or groups at high risk.

This research aimed to formulate a basis for strategic approaches for the prevention of dental caries in children based on the distribution of dental caries in the population at different caries severity levels. The objectives were to analyse the shapes of distributions and patterns of distribution of caries both within child populations and in individuals at differing severity levels and factors, such as presence or absence of water fluoridation, associated with the distributions. Using Rose's concepts on preventive strategies, approaches to the prevention of caries were developed based on the analyses.

The study was divided into three phases. First, both the incremental and distributive properties of dental caries were analysed using the longitudinal United States National Preventive Dentistry Demonstration Programme's data set. The results were subsequently tested using data from both the British Association for the Study of Community Dentistry's national programme in the United Kingdom and from a study carried out by the University of Wales and Walsall Health Authority. Secondly, the intra-oral distribution of caries, by tooth type and sites on the teeth, was analysed to provide the scientific basis for the identification of the differing components of a preventive package. The third phase developed the

findings from the first two phases to form the basis for strategy component selection at differing levels of caries.

Results indicate that standard relationships exist between the distribution of dental caries within populations and in individuals and that the risk of caries increments will affect the strategic approach. Knowing the DMF provides information on the prevalence and frequency distribution of caries, the variance, the teeth affected and the sites on the affected teeth that will be carious. There is a relationship between the mean caries score of a population and the prevalence of caries within a population which is independent of water fluoride levels. Furthermore, the distributive properties indicate that a small decrease in the risk for a whole population has a greater overall impact on total caries increment than a large decrease in high risk individuals. There is a hierarchy of susceptibility to caries within the mouth which is tooth and tooth site specific. The hierarchy is not linear, certain sites are grouped. This size of the grouping varies. At low levels of caries the groupings are smaller than at high levels of disease. A reduction in the attack intensity which benefited the groupings at higher levels of disease would lead to substantial savings in cavitated sites.

The findings suggest that the adoption of a policy for prevention should be determined by the caries level within the child population and that the hierarchical development should affect the choice of components for any preventive strategy.

At low levels of caries, only a relatively low percentage of people would benefit from a population based fissure sealant strategy, whilst at high disease levels substantial numbers of a given population will develop approximal lesions in those teeth which would be sealed. Fluoride reduces the overall attack intensity and is not site type specific in its action. Current shortfalls in knowledge relating to fluoride regimes prevent their impact from being modelled accurately.

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1: INTRODUCTION

1.1: Overview

Since the mid-1970's many industrialised countries have reported a decline in the prevalence of dental caries (Downer, 1984). Although the beneficial aspects of fluoride had been recognised for some time, and the use of a piped water system made delivery on a population basis a feasible option, acceptance of such programmes was limited. It was not until the development of an alternative fluoride delivery system which gave the consumers a greater ability to choose whether or not to use a fluoride agent did the decline in caries commence. Current evidence suggests that the single most important factor was the introduction of fluoride containing toothpastes (Rolla et al., 1991). The increase in sales of fluoridated toothpastes strongly correlates with the decrease in caries prevalence.

With the identification of the effects of fluoride on caries, other fluoride containing vehicles were developed to increase its availability. The usage of the agents was uncoordinated and little discussion occurred as to the possible implications of excessive exposure to fluorides (Szpunar and Burt, 1990).

In addition to the decline in the prevalence of dental caries the overall severity of attack decreased. The number of tooth sites affected by caries declined. This decline however did not have the same impact on all sites. The impact of fluoride on the caries process appeared to be unequal: certain sites appeared to remain susceptible. In particular, the relative proportion of lesions for large numbers of people became confined to pit and fissured surfaces. With the development of materials that could adhere either chemically or physically to pit or fissured enamel surfaces came the possibility of reducing levels of dental caries even further. However, the adoption of sealants by the dental profession was

haphazard and uncoordinated; many failing to use them, whilst others placed them on every pit or fissured surface.

In addition, little attention was paid to methods of addressing the cause of dental caries, the high consumption of sugars. The ease in which the decline in caries had occurred without any major efforts to reduce sugars consumption made the necessity to develop this aspect of caries preventive strategies a low priority. When combined with the likely difficulties that efforts in this area would face, programs aimed at reducing sugars consumption have played a relatively minor role.

With more and more individuals entering adulthood with fewer or even no carious teeth and a growing concern over the costs of dental care, the idea of targeting resources was postulated: the risk approach, (Stamm et al., 1988). By screening susceptible individuals prevention could be given to those who would benefit most. This gave rise to a choice in how preventative agents could be applied; to all, the population approach, to individuals, the high risk approach, or a combination, the directed population approach in which groups are targeted. The arguments put forward for alternatives to the population approach, as with the usage of fluorides, have to date paid little attention to the costs incurred when compared to benefits gained. The situation that has developed was of uncoordinated caries preventive strategies which have a very limited scientific basis. Little attention was paid to the economic costs of prevention and whether the strategies adopted were having an impact that could justify the expenditure. But perhaps most importantly, the public health aspects of strategy development had been ignored.

This project aims to provide a scientific basis on which to formulate caries preventative strategies. It will consider how the epidemiology of dental caries can

be used to develop appropriate preventive strategies, both in terms of the approach and its contents. In particular the project will tackle three issues: how the distribution of caries within a population changes at differing levels of caries prevalence and severity, secondly, how the intra-oral distribution of caries alters at differing severity levels, and thirdly, how the results obtained in the first two sections, can provide a scientific basis for strategy selection. It is hoped that the results will be used to help policy makers make rational decisions about how to tackle what still remains a major public health problem.

1.2: Aims and objectives

The aim of this study is to develop, test and apply a model that can be used to help plan preventive programmes for dental caries. Each stage of the study requires objectives to be set. The first is the development of the model. This requires a number of hypotheses to be tested. These are:

a. that there is a relationship between the overall mean caries experience and the prevalence of the disease;

b. that there is a relationship between the mean caries score and the associated variance;

c. that there is a relationship between the mean score and the percentage distribution at each DMF score; and

d. that a hierarchy of caries attack exists. At each DMF score the teeth and sites that have been attacked are known.

The second objective of the study is the verification of the model. To do that the parameters established for the model will be fitted to other data and comparisons made.

The third objective is to apply the model to differing caries scenarios to provide a scientific basis for the formulation of strategy approaches in the prevention of dental caries in children.

2: REVIEW OF THE LITERATURE

2.1: Introduction

There are a considerable number of choices to be made by policy makers when considering dealing with ill health. These choices occur both among the varying health sectors, for example within the community sector between chiropody and dental care, and between what balance of prevention and treatment a health problem should be tackled with. Resources are finite, health care is competing with other areas such as education and social services for funding. Decisions as to the most appropriate use of resources must be made and the choice cannot occur in a financial vacuum. If finance is given in one area, another area will do with less. Whilst the basis of strategy selection should be correct scientific knowledge it will also involve political decisions. Policy making is a dynamic process in which strategy selection is one component. Abel-Smith (1996) identifies three forces affecting policy making within the health sector. First there are demographic changes. For example, if there is an increase in the elderly population, one of the largest consumers of health resources, one would expect a change in the resource allocation to follow suit. Second, changes in medical technology. These invariably lead to higher costs. Third, there are growing public expectations. It is the third of these forces that Abel-Smith sees as being consequential in forcing policy makers to concentrate more than ever on the need to deploy resources efficiently and effectively. These forces can create a situation in which, despite having the knowledge to prevent or treat a disease, the political will is not there. Thus, when considering the appropriate strategy for dealing with ill-health careful thought should be given to the effects that the policy maker is trying to achieve. In an ideal situation, if the knowledge to eradicate a disease exists, the political will necessary to achieve the goal should also be present.

In accepting that a political component in strategy selection exists however does not detract from its scientific formulation. Any strategy must be based upon sound scientific knowledge. In addition economic measures should be utilised to help justify strategy selection. Scheffler and Paringer (1980) in a review of the economic evidence on prevention, divided preventive strategies into three categories: lifestyle changes, public health measures and screening programmes. Caries preventive strategies have components associated with all three categories. The most important of the aspects is the change in lifestyle resulting in a reduced sugars consumption. Sugars consumption is recognised as the principal cause of dental caries (Committee on Medical Aspects of Food Policy, 1989). A reduction in sugars consumption is the only true preventative measure. The public health measure would, if appropriate, include water fluoridation, and in the final category, a screening programme for dental caries.

For prevention to have a sound economic argument for its adoption disease must be present. It does not make economic sense to expend resources to prevent a disease which does not exist. In addition it is not only economic benefits that need to be studied when analysing the appropriate strategy. The non-economic benefits, such as a reduction in pain and suffering, may be considerable. Furthermore to enable the appropriate choice to be made, in addition to the benefits, the risks of adopting a particular strategy must be considered along with the costs. While tooth loss is primarily due to caries, orthodontic extractions in the younger age groups and periodontal disease in older age groups can give rise to loss of teeth. Should there be a decrease following adoption of a strategy in both tooth morbidity and mortality due to caries, any increase due to orthodontic problems or periodontal disease would be an undesirable outcome. Would a caries preventive strategy, reducing pain and suffering due to a pathological

process be more appropriate if it gives rise to mental anguish through unacceptable crowding?

Consideration needs also to be given to other options. For example, at a given level of disease, the costs involved in the treatment of the disease may be lower then for preventing it. Alternatively, a society may be willing to accept a level of pain and suffering in return for other benefits obtained by the use of the resources in a different field.

These issues raise a number of questions that should be considered when planning a caries preventive strategy. What are the measurements that policy makers can utilise to help make choices? Is there a level of disease for which treatment is the more appropriate policy, and if so, what is the level? What additional considerations should be taken into account when considering costs other than economic ones? Finally, with teeth being lost for other reasons than dental caries, what increase, if any, would occur in these other disease processes which may negate the costs of the caries preventive strategy?

Oral health care is not alone in health care when considering these issues. The problem of outcome measurement continues to be at the forefront of the policy making agenda. No longer will mortality rates suffice as a measurement of the failings or successes of a health care system.

In oral health care the standard measure of caries experience is the DMF index. This, however, is a measure of past caries experience and it is not reversible. A person with a particular DMF score cannot revert to a lower DMF score. In addition equal weighting is given to the components of the index. A DMF of 1 might be a decayed score, a filled score or a missing score, yet the implications for an individual are entirely different. Furthermore little account is made for an

individual's age. A 6 year-old with a DMF of 5 will have differing planning implications than a 65 year-old with a DMF of 5.

This review of the literature concerning strategy selection is divided into three sections. In the first, the literature concerning the patterns of distribution of dental caries is reviewed. In the second part the scientific basis for strategy selection for the prevention of dental caries is reviewed. This section covers both the approaches and the components that can be used in a dental caries preventive strategy, namely diet, fluorides, fissure sealants and oral hygiene. Finally, a review of the literature on the principles of health economics and their application to the prevention of dental caries is provided.

2.2: Issues in Strategy Selection

2.2.1: The Epidemiology of dental caries

The existence of a strategy implies that there is an organised plan to reach a goal. Knowledge of having achieved the goal of any strategy in the most efficient manner requires an understanding of the disease process. For the prevention of dental caries this includes not only the natural history of a carious lesion, but how the disease presents itself both within the mouth and at a population level: are there patterns to the distribution of dental caries? The question that subsequently arises is: are there situations in which certain resources should be utilised rather than others: does the problem of dental caries present in differing states?

The distribution of dental caries is crucial to the understanding of the nature of the disease process. It is necessary to establish both the cause, and subsequently, to

prevent the disease. The first step to establish the cause is to make an assessment of the magnitude of the problem. This needs an index of measurement. Since 1938 the commonly used index of dental caries is the DMF index (Dean et al., 1942). The DMF index measures the severity of disease. When calculating DMF data for a population the average scores are calculated and the values compared. This, however, makes two assumptions. First, that the distribution of the disease is normal, thus allowing the use of parametric statistics. Second, and perhaps more importantly, that the scale of measurement is linear. A change of the same magnitude at any point on the scale is a constant. To date the assessment of the effectiveness of any caries preventative measure has made these two assumptions. These assumptions need to be validated in order to formulate appropriate dental caries preventive strategies. If these assumptions are wrong, current methods in evaluating the effectiveness of methods could be incorrect. This review of the distribution of the dental caries will provide the background to the question on whether the two assumptions are valid, and as a consequence, the implications this may have for assessing any preventive measure.

2.2.1.1: The distribution of dental caries

One of the first detailed reviews of the epidemiology of dental caries was published in 1952 (Finn, 1952). In the section entitled "The prevalence of dental caries" Finn reviewed available data and drew a number of conclusions, the majority of which are still valid. In his summary Finn concluded:

"Dental caries in individual teeth increases in both the number and the size of the lesions proportionately to the number of years of exposure of these teeth to the oral environment. There is a difference in attack rate for the morphological types of teeth"

In addition, the prevalence of dental caries has varied considerably through history. Certain groups appear to be have been more prone to dental caries than others. For example, Stone Age man had a lower prevalence of dental caries when compared to the population of Great Britain in Edwardian times (Hardwick, 1960). Furthermore, as the prevalence of dental caries changed, so did the extent of individual lesions. At low prevalence levels the most common form of cavitation was the occlusal cavity. As the prevalence rose the type of lesion involved approximal and smooth surfaces. This suggests that certain sites are more prone to cavitation than others. The evidence for these patterns is presented below and possible implications drawn.

Major differences exist between the average dental caries experience of nearly all groups prior to the 19th century when compared to groups studied after this time. In addition, certain sections of populations today, and in particular those who have not adopted a Westernised diet and may have been exposed to fluoride, have a remarkably similar prevalence of dental caries when compared to populations prior to the 19th century. In these populations the prevalence and the severity of dental caries is very low, although some important differences may exist in the intra oral pattern of dental caries. Lenhossek (1919), Szabo (1935), Krikos (1935), and Whittaker et al. (1981) all drew the conclusion that ancient man was almost completely free of dental caries. Furthermore, where dental caries was found, it tended to be in the older age groups. Leigh (1930) and Marston (1936), again suggest that major differences exist between the prevalence seen in the 1970's and those of Stone Age man. More recent studies by Moore and Corbett (1971; 1973), Lunt (1972; 1974), and Kerr et al. (1988; 1990) reinforce these ideas. Von der Fehr (1986), with an overview of dental caries prevalence throughout history, drew attention to the rapid change during the 19th Century. This overview, along with the findings of the studies by Kerr et al. (1988; 1990)

also drew attention to the site specificity of the dental caries attack pattern. Von der Fehr noted that with the increase in prevalence, front teeth caries became very common. Kerr et al. (1988; 1990) drew the conclusions that the pattern of prevalence and incidence of dental caries attack is age dependant. These conclusions have been reinforced by a number of authors.

However it is important to recognise what is meant by dental caries. Mandel (1990) defines it by stating that dental caries is a process. There will be forces directing the process in one direction, and other forces opposing this. As such the dental caries attack is not age dependant necessarily but time dependant. The rate at which caries occurs is dependant upon the strength of attacking force and the resisting forces. The resisting forces may be structural, non fissured surfaces against fissured surfaces; mechanical, fissures sealants acting as a barrier; or chemical, with fluoride altering the environment in which the challenge is occurring. The impact of mechanical and chemical mechanisms are as likely to give rise to variation within a population as genetics: their impact being dependant upon a number of factors including operator sensitivity and the advancement of technology. The changes in the epidemiology of dental caries are in consequence not necessarily related to changes in the structure of teeth but due to the conditions that alter the force of the dental caries attack. That is, the pattern of dental caries in a 14 year-old from Western Europe in the 1970's could be the same for a 50 year-old Stone Age man for a given DMF. McDonald and Sheiham (1992) raise this issue. They suggest that the dental caries attack pattern is independent of fluoride. Against this, certain authors have made the suggestion that fluoride has a greater preventive role on smooth and approximal than occlusal surfaces (Backer Dirks et al., 1961b; Horowitz et al., 1972; Ripa et al., 1985). By examining the methodological issues and the distribution of dental caries attacks within the oral cavity the next section will examine the evidence for this suggestion.

2.2.1.2: The pattern of dental caries

The word pattern means that a structure has a logical form. This implies that, in order to compare a pattern, the components that give rise to a shape can be measured. In dental caries those components can be defined both by the different morphological characteristics of the teeth or tooth surfaces and the number affected.

In oral epidemiology when studying the distribution of dental caries, the surface is the smallest unit of measurement used. Tooth surfaces can be split into 3 categories, smooth, approximal and occlusal. The 32 adult teeth have a total number of 148 surfaces. These can be split into the following categories: 20 occlusal, 64 approximal, and 64 smooth surfaces. Due to the morphology of certain surfaces, namely the buccal aspects of the lower first permanent molars and the lingual aspects of the upper first permanent molars, a more appropriate categorisation would be pit and fissure as opposed to occlusal. Indeed given the selective effects of one group of preventive agents, fissure sealants, this would be a more sensible classification. The question that faces the researcher is "has the proportion of each type of tooth surface altered for any given DMF-S?" This question gives rise to a hypothesis that can be statistically tested, "what is the probability of any given tooth surface undergoing a carious attack at a given DMF-S?"

However, it is important to recognise that the pattern of dental caries can be examined in two differing dimensions: the severity dimension and the progression dimension. First, the severity dimension. For a given DMF, which are the sites which are involved? For example, at a DMF-S of 2, are certain sites more likely to be found carious than others, and furthermore does this vary with the presence of fluoride, age or other variables? The second dimension is that of caries

development. As individuals increase their DMF score is there a logical development? Can one predict which sites or teeth will subsequently be affected? The two issues, although linked, are different. An individual with a DMF-S of 2 has a differing number of components in the pattern when compared to an individual with a DMF-S of 3. One cannot compare differing DMF-S values to assess the patterns in the first dimension only in the second, on how the severity of dental caries attack develops. In essence, is there a hierarchy of attack?

2.2.1.3: The hierarchy of attack

The literature on patterns of dental caries would suggest that a hierarchy of dental caries exists. In 1874, Parker (1874) reported that there was an order of susceptibility of the permanent dentition to dental caries. The findings, covering the age range of below 8 to over 65, only covered the upper dentition. Parker suggested that the first molar was the most susceptible followed by the second molar. The next most susceptible tooth was the second premolar followed by the upper lateral and then the upper central. These were followed by the first premolar, the third molar and the least susceptible, the canine. In reviewing the literature on individual tooth susceptibility, Finn (1952) identified 11 studies, including that of Parker, prior to 1939. The findings of all of these studies were very similar. The most susceptible teeth were the first molars, with the lower molars cited more frequently than the uppers. The least susceptible teeth were the canines, although a number suggested that the third molars were surprisingly resistant given their morphology. One possible explanation is that, at the time of the examination and given that dental caries is a process, the third molars had not yet been in the mouth for sufficient time to be classified as decayed. This raises an important methodological issue is answering the question as to whether the development of dental caries follows a pattern. The decay process can start as

soon as a tooth erupts into the oral cavity. One could therefore hypothesis that the order of susceptibility of teeth is the same as their eruption times. Whilst this holds for the first molar, normally the first tooth to erupt, the subsequent susceptibility patterns does not relate to tooth age. One example is the variation in dental caries prevalence found between the second molar and first premolar.

Both the second molar and premolar teeth have fissured occlusal surfaces and the distal aspect of the second premolar and mesial aspect of the second molar will provide approximal contact with the first molar tooth. Their eruption times are also similar, yet the prevalence of dental caries is far greater in the molar tooth when compared to the premolar. While this does not exclude the possibility that one determinant of susceptibility lies in the morphology of the tooth, other factors suggest another hypothesis. For individual teeth, it is not only the morphology, and in particular the presence or absence of a pit or fissured surface that determines a tooth's susceptibility but, in addition, the position of each tooth within the oral cavity also contributes to the susceptibility pattern.

Knutson (1958) first suggested that there was a relationship between the caries prevalence of a population and the severity of the attack. Massler et al. (1954) developed this argument to set up a 'channel' system. Based on annual incremental data they proposed the hypothesis that the level of severity of the caries attack, if known at certain ages would allow the 'prediction' of dental caries susceptible groups. The groups with the higher severity level at 6 years of age would follow the higher 'channel' or track representing the higher dental caries increment. The allocation to the 'channel' was again based on a hierarchical methodology. If the lower first molars were only involved at 8 years of age, the attack was said to be mild. If, at the same age, all four first molars were affected, the attack was said to be moderate. The idea of individuals residing in susceptibility 'channels' was also highlighted by Carlos and Gittlesohn (1965). In

the analyses of the data from several studies, they concluded that an exponential relationship existed for susceptibilities.

Akpata and Jackson (1978) examining urban Nigerians found that the second molar appeared to be more vulnerable than the first molar. This view was echoed by Jensen et al. (1973), and Westwater (1977). Both studies found a higher dental caries prevalence in the second molar than the first. It is interesting to note that all three studies were in Africa and that the age of the populations examined were approximately 12 years old. Cleaton-Jones and Walker (1980) reported that dental caries prevalence of the second molar was three times that of the first molar in black rural schoolchildren when compared to white children, yet equally prevalent in the teeth of urban children. There are a number of possible reasons for this apparent difference when compared to findings from other populations. One possible explanation was suggested by Manji et al. (1986). As the development of dental caries is a process, should the process be sufficiently slow, the occlusal surface may be ground down by attrition and abrasion. As the second molar will only just have erupted, the occlusal surface of these teeth are unlikely to have been in contact with the opposing jaw to allow a 'natural' wearing to take place. Thus, while the wearing action will have occurred in the first molar occlusal surfaces, and hence they will appear to be free of dental caries, the occlusal surfaces of the second molar will have a level of dental caries attack. This hypothesis is also supported by the work by Kerr et al. (1988; 1990).

The pattern of dental caries attack found in the Scottish Medieval population was different than is currently the case. The dental caries attack tended to occur at the cemento-enamel junction. The hypothesis was put forward that, due to severe occlusal wear, the teeth underwent compensatory eruption and the subsequently exposed root surface was more prone to dental caries attack than the coronal surfaces.

A second, and perhaps more plausible reason, is that the tooth surface of the second molar may not yet be fully mature and that 'caries' like lesions could have been recorded. Frencken et al. (1989) again in Africa, examining Tanzanian 12 year-olds, but using different criteria with a graded level of the D component, found that the first molar was more prone than the second, as did Keruso et al. (1986) and a further study by Manji et al. (1989). These differences could well be due to a change in the dental caries attack force. This hypothesis is supported in that the order of susceptibility was different only in rural populations whose diet was more abrasive.

Nevertheless, in all of the studies cited the occlusal surface was more prone to dental caries attack than either the smooth or approximal surfaces. Indeed, Eklund and Ismail (1986) stated that occlusal caries precedes all other types, and increases most rapidly and to the highest levels in the molars. Furthermore they suggested that the decline that has occurred in many Western Industrialised countries is probably more a delay that true primary prevention. The strength of the dental caries attack may affect the site specific presentation within the mouth. At very low levels of attack, where other mechanisms such as occlusal wear may influence the caries process, a different pattern to that found in most situations will exist. In all of the surveys mentioned, where the attack pattern appears to differ from the normal, the overall dental caries prevalence is very low.

Interestingly the pattern found in the deciduous dentition suggests that other factors may influence susceptibility. The position within the mouth appears to be a more likely determinant of dental caries susceptibility than either the eruption time or surface morphology. Both Colditz (1931) and Mellanby (1934) suggested that the second deciduous molar was the most susceptible tooth. More recently Ripa et al. (1985) reported that in the deciduous dentition smooth surface

involvement to be as likely as fissured surfaces. This suggests that any hypothesis treating the oral cavity as a single unit is too simplistic. In addition to the possibility that tooth surface morphology plays a role in the determination of the dental caries attack pattern, the anatomical, bacteriological, and physiological aspects of varying sites within the oral cavity are also crucial. These possibilities were outlined by Weatherall et al. (1989; 1989) and Primosch (1986) with specific reference to both the retention of glucose and the clearance rate of fluoride from varying sites within the oral cavity.

More detailed analyses on site specific data have been reported by a number of authors. Marthaler (1966) suggested that certain occlusal surfaces were more susceptible than approximal or smooth surfaces. He went on to suggest an alternative hierarchical method of assessing dental caries severity (Marthaler, 1972). This ranking was said to be simpler whilst retaining accuracy. In consequence it would save time when compared to the full mouth examination. The idea that the mouth could be zoned, so enabling a more efficient data collection method, was also suggested by Grainger (1967), Viegas (1969) and later Poulsen and Horowitz (1974). In addition to zoning Hadjimarkos (1967), using half mouth examinations, and more recently the examination technique employed in the national survey of The Netherlands (Truin et al., 1991), both adopted a partial mouth examination system for data collection. Inherent in these methodologies is the idea of symmetry.

Dental caries when measured at a cavitation level, is as likely to occur on the left hand side as the right hand side of the mouth. A number of authors have examined the idea of symmetry. Walsh and Smart (1948), examining the relative susceptibilities of differing tooth surfaces in New Zealand, suggested the existence of symmetry. Using cross-sectional data Jackson et al. in a series of papers (Jackson and Burch, 1968; Jackson and Burch, 1970; Jackson and Burch,

1972; Jackson et al., 1972) examined the distribution of dental caries in anterior teeth. The findings supported the idea that dental caries is both non-random in distribution, that is certain sites are more susceptible, and that there was a left-right symmetry.

A study by Murray et al. (1976) also examined patterns of dental caries in incisor teeth and highlighted the surface specificity of the attack. In the study reference was made to a step like progression which occurred for even numbers of sites affected. This adds further evidence to the suggestion that, once the potential for a site to cavitate has been reached on one tooth, it has also been reached on at least the contra-lateral tooth. The potential may also have been reached on other teeth. In studies of the bilaterality of dental caries Berman (1970), Wood(1985), DeJong and Dunning (1971), Rizak and Razak (1988), and Houjoel et al. (1994) all suggested prevalence symmetry, but not necessarily of homologous pairs. Again, in the statistical methodology applied, the assumption was made that the potential for homologous pairs was the same and that it was specific for those sites. This ignores the previous point that, should the potential have also been reached on other teeth, the prevalence symmetry would continue to exist but it could appear that the homologous paired symmetry had been lost. However, the literature does suggest that the susceptibility of a site is dependant not only upon its position within the oral cavity but its morphology.

A further complication in any assessment of susceptibility is that enamel may well undergo a period of maturity (Kotsanos and Darling, 1991). During this maturation period the tooth surface will become more resistant to dental caries attack. Thus changes in the attack rate need to be studied. A study by Johnson and Gjermo (1989) came to the conclusion that the attack pattern was independent of oral hygiene, sugars consumption and knowledge level and that enamel maturation was a key component. Besides the aforementioned longitudinal studies

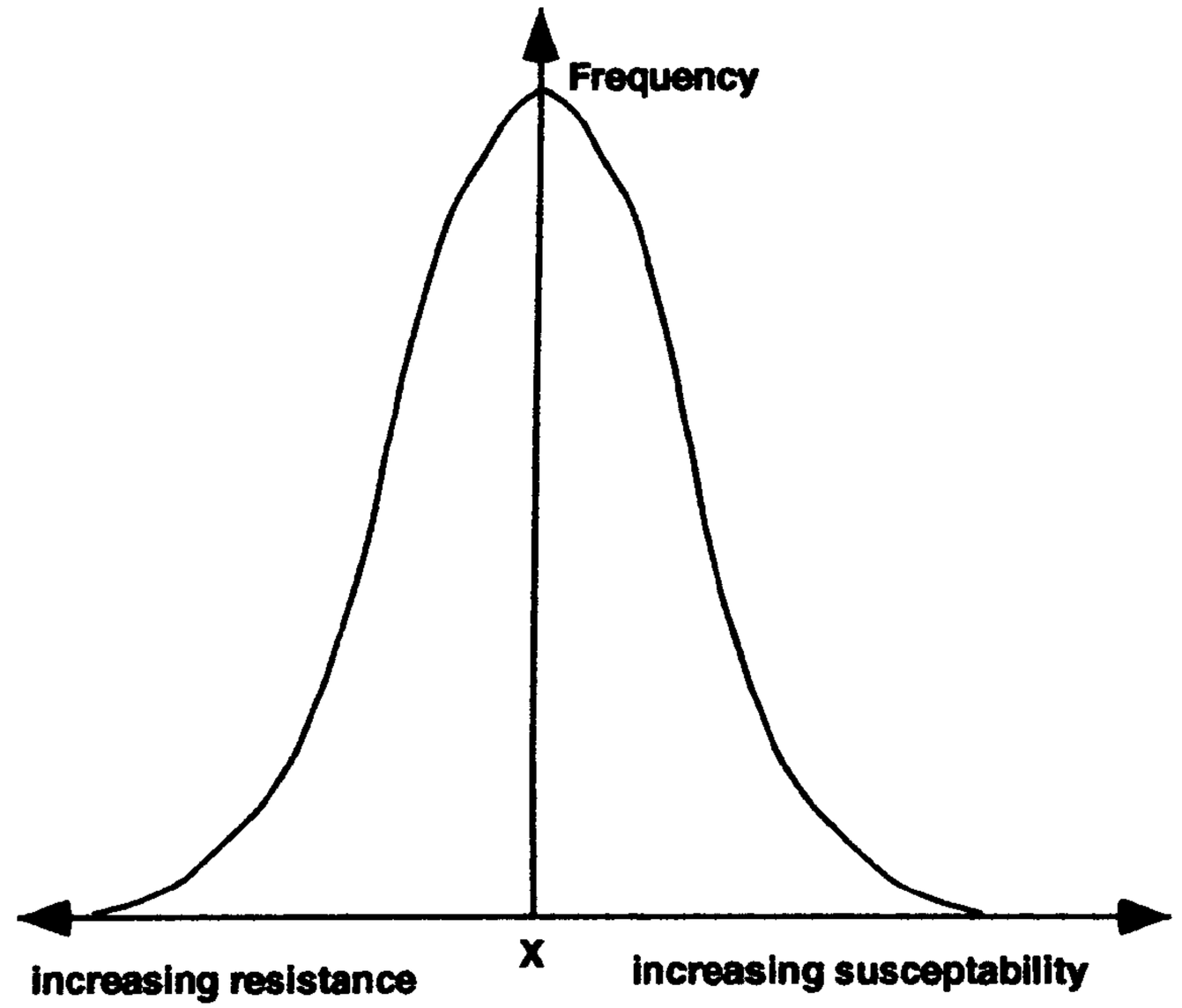
on anterior teeth, there is also a growing volume of data concerning caries distribution in posterior teeth, particularly the first permanent molar tooth (Wood, 1985; Mansson, 1977; Carvalho et al., 1992). Indeed it is prudent to point out that as the overall prevalence of dental caries declines, the one tooth which still suffers from a relatively high prevalence of dental caries is the first permanent molar. Kolmakow et al. (1988) examined three age groups and compared the status of the occlusal surfaces to the smooth surfaces with reference to the usage of fissure sealants. Using a relative risk model, those individuals with a dental caries attack were far more likely to have other surfaces undergoing an attack when compared to the group whose occlusal surfaces remained dental caries free.

Dummer et al. (1990), examining the changes in the distribution of decayed and filled surfaces, also reported the relative increase in occlusal caries with respect to other surface types. This was also the conclusion of Burt (1985a) in a review of the changes in American schoolchildren. Vehkalahti et al. (1991) reported on the decline of dental caries attack over a ten-year period in Helsinki. In addition to the decline in overall prevalence, they found that the decline had occurred for both smooth and pit and fissure surfaces though not to the same extent; the decline in smooth surfaces being greater. Edward et al. (1992), examining two cohorts of children found that, coinciding with the decline, that differences between certain sites had also declined. A more detailed distribution of surface-type dental caries attack was presented by Kumar et al. (1989). One important finding was that, although there remained differences in dental caries experience attributable to the socio-economic status, as the prevalence of dental caries had declined, so had the magnitude of the differences. These differences however were of differing magnitudes dependant upon the surface type. This has lead to the suggestion that fluoride has a more pronounced preventive role on smooth and approximal surfaces than pit and fissure surfaces.

The longitudinal pattern of development on occlusal surfaces of the first permanent molar over an eight-year period was made by Vehkalahti et al. (1991). They came to the conclusion that the dental caries attack was most frequent during the age range 7 to 9 years, that is between 1 and 3 years after eruption. East (1941) suggested that a negative relationship between tooth eruption time and susceptibility existed; the longer the teeth were in the oral cavity the less likely they were to undergo dental caries attack. Fukada et al. (1982) and Miyano et al. (1974) both reported that the rate at which dental caries attacks occur is dependant upon the time since eruption, the most frequent attack occurring immediately post eruption. Ripa et al. (1985) came to different conclusions suggesting that the time that the teeth were in the mouth had little effect on the vulnerability of occlusal surfaces to attack. This study examined children in whom the molar teeth had already been erupted for, perhaps, up to 5 years. King et al. (1980) and Bohannan (1983) both reported a sustained dental caries attack rate that neither proves nor disproves the hypothesis that a tooth surface is more susceptible immediately after eruption. However it is prudent to point out that within a population, that for a given attacking force, one would expect to find a normal distribution of susceptibility. A variation within populations of resistance factors will exist. Certain individuals will have a higher resistance to caries than others. Hence, as Ripa points out, the attacking force will be the determinant of the rate at which new dental caries lesions appear.

This can be explained in statistical terms as follows. A population would have a distribution of susceptibilities which is normal in shape. The horizontal (x) axis represents the susceptibility, the mean value is set at X. Those individuals to the right of the origin represent the members of the group who have a greater susceptibility than the average, Figure 1. For a given attacking force, they will experience dental caries before the majority. To the left of the origin are a group who are less susceptible, that is appear more resistant to a dental caries attack. At

Figure 1: Distribution of caries susceptabilities for a given tooth site within a population



a high levels of attack, as the force would be sufficient to overcome a greater resistance factor, the variations of susceptibility in individuals would become less. It would thus appear that the rate of attack occurred most frequently immediately after eruption. The only time that the attack rate was independent of eruption time was at a very low level thus allowing the monitoring of variations in rates.

The paper by McDonald and Sheiham (1992) takes this argument a step further. The proposal was made that there was a relationship between vulnerability of differing sites and overall dental caries severity. Furthermore this was independent of fluoride. This has enormous implications, not least for the usage of fluoride as a preventive measure. There remains however a major flaw in this work which lies in the statistical methodology used. In the paper it is assumed, wrongly, that the probability of any surface becoming carious is a constant. However, once a surface has become carious, that surface can no longer be influential in determining the future pattern of development of caries: it cannot revert to a non-carious situation and undergo an attack. Hence the site should be excluded from future analyses. For example, suppose there were 4 surfaces. The probability of any one of those surfaces undergoing an attack initially to give a DMF score of 1 is 1:4 or 0.25. Subsequently, the probability of the development of next carious lesion to give a DMF score of 2 has changed. It is no longer 0.25 but 1:3, that is 0.33. Indeed the probability of the final surface of the four becoming carious next is 1: no other surface can do so. Thus the curves presented by McDonald and Sheiham, apart from the first lesion, do not represent independent variables, and in consequence they must be related. Despite this methodological flaw, the fact that the curves are similar for differing levels of fluoride exposure, must not be ignored when considering the possible impact of preventive measures.

2.2.2: The Scientific Basis for the Prevention of Dental Caries

A strategy was defined in Section 2.2.1 as '..an organised plan to reach a goal'. Any plan has a number of components of which the contents and the usage of those contents should be described. In the formulation of a dental caries preventive strategy, both the components and their appropriate usage should be described. First, content.

The scientific evidence highlights three major and one questionable minor component. The first component is diet, the principal aim being to reduce the exposure to non-milk extrinsic sugars (Committee on Medical Aspects of Food Policy, 1989). The rationale behind this component is to reduce the nutrient source for the oral micro flora, thus reducing the volume of acid produced for a given time. The second component is fluoride. Current thinking suggests that fluoride acts in a number of ways such as aiding remineralisation of enamel, affecting plaque, and inhibiting acid production and that it can act through both topical and systemic mechanisms (Margolis and Moreno, 1990). Third, fissure sealants. Their role is to increase the resistance of certain tooth sites to demineralisation by providing a mechanical barrier to acid thus preventing demineralisation. Tentative evidence also suggests that a fourth component, oral hygiene, may play a direct role. However the importance of this component may have been over-stressed. The evidence for each of these components is examined in the first part of the review.

The second aspect of strategy selection is the appropriate usage of the agents. This statement implies that different components work more efficiently and effectively in differing situations. For example, Stamm et al. (1984) suggested that topical fluorides in certain situations, whilst being effective, are far from

efficient and they are of little value as part of a preventive package. Furthermore, fissure sealants are effectively only on certain sites. If, for a given level of dental caries, the prior application of fissure sealants would fail to reduce the level of disease, their usage would be inappropriate. Thus knowledge, not only about the effectiveness of agents at differing levels of dental caries, but of the patterns of dental caries are important. These issues are discussed in the second part of the review.

2.2.2.1: The content of caries preventive strategies

When considering how to reduce levels of dental caries the literature produces unequivocal results. A reduction in sugars consumption, exposure to fluoride containing products and the application of fissure sealants all impact.

First sugars. A number of authors have summarised the relationship between sugars and dental caries, (Sreebny, 1982; Sheiham, 1991; Burt, 1993), with Shieham postulating precise measurements for the exposure required to avoid caries. However few authors have examined the relationship using a cohort methodology, possibly due to the inherent difficulties with the methodologies. There have been two major longitudinal studies looking at the relationship between dental caries increments and sugars consumption. Rugg-Gunn et al. (1984) examined 405 11-12 year old children over a 2 year period. Although the correlations between the dental caries increments and dietary factors were small this was in part explained by the overall low caries increments observed. Nevertheless there was strong statistical evidence for a relationship between sugars consumption and dental caries increments when the highest and lowest sugars consuming children were compared. The second study was carried out by Burt and Spuznar (1994). Again poor relationships, in terms of correlation

coefficients, were found for sugars and dental caries increments. However, as with Rugg-Gunn et al., the group identified by their high sugars consumption also were at a greater risk of having a higher caries increment when compared to the group with a low sugars consumption. These results highlight the arguments put forward by Rose (1993) which are discussed in the section concerning strategy approaches.

The second component, fluoride, also should be considered with respect to the dose response curve as highlighted by Dean et al. (1942). The important issue in this study is that the results were obtained for an assumed water intake. What the data do not show is what the actual water intakes were. This is important as, at a lower level of water intake, to have the same effect on dental caries rates, a higher concentration of fluoride would be required when compared to a population with a higher water intake. It is not the fact that one part per million is the appropriate level, the question is: how much fluoride is necessary to give the optimum preventative effect given the current level of fluoride that the population is exposed to? This issue was highlighted in the study by the findings following the cessation of artificial fluoridation in the Netherlands (Kwant et al., 1973).

The trial, initiated before the advent of other population fluoride vehicles, was set up to examine the impact of artificial fluoridation in one area, Tiel, with a control non-fluoridated area, Culembourg, in the Netherlands. With the introduction of fluoride into the water, a sharp decline in caries levels occurred in Tiel. While Culembourg also experienced a drop in caries prevalence, levels in the area always remained higher. These results were reported after various time spans following the introduction of fluoride. Backer Dirks et al. (1961a) reported results from the study over the first six and a half years and Kwant et al. (1973) subsequently followed on. With the cessation of artificial water fluoridation there was an increase in dental caries severity in Tiel, but not to the pre-fluoridation

levels. Indeed the current studies indicate that the decline in dental caries severity has continued in both areas, with a reduction in the size of the difference between the two towns. It should be noted that the commencement of the trials occurred before widespread use of other fluoride containing vehicles. Similar findings occurred in a study by Thylstrup et al. (1982) in Denmark. The Danish Child Dental Health services have adopted a school based fluoride rinsing programme since 1966. With the widespread use of fluoride containing toothpastes the majority of children were being exposed to two fluoride agents. When comparing children in areas where the water supply was sub-optimal to an area with optimum levels, the differences in DMF-S data were negligible.

As such it would appear that all fluoride trials, irrespective of the vehicles involved suffer from one fault. The position that the study populations are on the fluoride dose-response curve is unknown. What is being assessed in the trial is the fluoride agent plus any background level. It is the contribution that any agent under study makes to the total exposure to fluoride that will determine its impact. The impact of a fluoride agent on a population already exposed to a high level of fluoride when compared to a population with a low level of exposure will be very different: the benefits to the low exposed group are likely to be greater. Without assessing the existing fluoride exposure before undertaking a fluoride trial, comparison between it and other fluoride trials has a shortcoming: one may not be comparing like with like.

The overall picture for the use of fluorides would indicate that no one agent is able to provide the optimum level (Burt, 1989). Whilst fluoridation of water appears to have been the most effective, the value of fluoridated toothpastes should not be underestimated (Rolla et al., 1991; Davies et al., 1995). The overall picture is one in which fluoride exposure appears to have been the major factor in the decline seen (Petersson and Bratthall, 1996). The issue of compliance needs to be

mentioned. In public health terms the advantages of water fluoridation include that it offers an equity in terms of coverage, and little or no compliance is necessary. Toothpaste suffers from a compliance problem: individuals need to be able to afford and brush their teeth using it. Against this, it has the advantage that its usage can be linked in with other oral health messages for mutual benefit, for example the reduction of plaque to help improve periodontal health.

The review of the contribution of fluoride in caries prevention highlights two major issues. First, as the decline in dental caries has occurred the contribution of the more specialised fluoride vehicles in caries preventive strategies has been challenged. Generally, the more compliance that an agent requires the less effective the measure is. Secondly, a missing component within the assessment of the effectiveness of fluoride regimes is the background level of fluoride existing when studies have been carried out. Both Dean et al. (1942) with water and Stephen et al.(1988) for toothpaste, have highlighted the dose response relationship between fluoride and caries. To assess the benefits that fluoride may contribute to a caries preventative strategy, the existing level of fluoride must be known.

2.2.2.2: The evidence for using fissure sealants as part in dental caries preventive strategy

It has been suggested that by combining the use of fluoride agents with fissure sealants dental caries can be virtually eradicated (Ripa et al., 1985a). With the decline in the prevalence of dental caries in many countries, and the associated increase in the proportion of sites that suffer from dental caries that are pit and fissure surfaces, the logic for their use when the caries situation is low would

appear to be sound. However the usage of sealants remains confined to pit and fissures; the morphology of these sites providing the basis for sealant retention.

Many authors have reviewed the literature on sealant restorations, including Ashkenazi et al. (1985), Ripa (1986), Stephen and Strang (1985), and Williams (1990), who concluded that a failure to use sealants "must constitute neglect". Extensive clinical trials, including a 10 year assessment (Wendt and Koch, 1988) indicate that fissure sealants are effective. However there appears to be a wide range of definitions of effectiveness (Weintraub, 1989). Some authors regard it to mean retention (Little, 1986), some partial retention, (Dorignac, 1987), others the failure of the surface to become carious (Burt et al., 1977). In addition, Weintraub (1989) highlights that others have simply reported the number of teeth or sites sealed and some simply the number of children. All of these shortcomings mean that any valid review of the effectiveness of fissure sealants is difficult.

In her review of the effectiveness of pit and fissure sealants Weintraub (1989) concluded that it was important to determine the length of time that the assessment was made over. The median effectiveness figure for fissure sealants varied not only according to the length of time of the trial, as one would expect, but also the fluoride status of the population. Over the first two years of a trial, the effectiveness of any programmes was greater in populations who received fluoridated water when compared to those in which the water was not fluoridated. This result however was reversed between the second and fourth years, after which the impact of fluoride status was negligible.

In her concluding remarks Weintraub suggested that targeting certain groups such as children and young adults is appropriate. The idea of targeting groups is not new, both the British Paedodontic Society (British Society of Paediatric Dentistry, 1993), American Dental Association (Council on Dental Health and

Health Planning and Council on Dental Materials, Instruments and Equipment, 1987), and more recently, the North York Group in Canada, (Woodward et al., 1995), have issued guidelines. Selection criteria include being handicapped, socially disadvantaged, and most commonly, past dental caries experience. Of particular note is the recommendation that, if one permanent molar tooth has occlusal caries, the three remaining first molars and all second molar occlusal surfaces should be sealed. This raises a key issue. By acknowledging that an individual with a DMF-S of 1 requires up to eight fissure sealants, one is also acknowledging that it is the most appropriate use of resources: this approach is superior to any differing approach. Should an individual, at present with a single lesion develop eight lesions, and all eight were confined to pit and fissured surfaces, the approach might be justified. However, should new carious lesions be confined to fewer teeth, the efficiency arguments would change. Certainly, the distribution of dental caries severity within many populations would suggest that this approach is wasteful and hence an inefficient use of resources.

What is apparent is the acceptance that, as part of a public health strategy, fissure sealants are unacceptable for the entire population. There may well be individuals in whom selected teeth can be sealed, and that this would depend upon the likely distribution of disease at differing ages.

In conclusion, the scientific evidence points to two objectives to a dental caries preventative strategy. The cause of dental caries is exposure to sugars. Irrespective of other steps taken, dental caries would continue to develop once the level of intake of sugars is above the region of 15-18Kg per person per year (Sheiham, 1991). Fluoride would appear to slow down the rate at which dental caries develops but it should be regarded as inferior to a sugars policy as, like fissure sealants, it fails to tackle the cause. However, given the practicalities, it may well be easier to reduce levels of dental caries by reaching the optimum

fluoride level than reducing sugars consumption. Both sugars and fluorides have a dose-response relationship with respect to dental caries increments. It would appear to be too simplistic to identify the reason for any change in dental caries prevalence without knowing the position on both dose response curves of a population. An increase or decrease in both agents of a relative magnitude on the dose response curves may not alter the dental caries increment. Addition of fluoride in whatever form would have no effect on the disease if the level available to the individual was already on the plateau. Thus, in order to formulate the appropriate strategy, the two pieces of information required are:

i. sugars consumption, and
ii. current level of fluoride exposure.

The evidence for the adoption of fissure sealants is far from conclusive. While in terms of clinical effectiveness they have a strong case, their net effectiveness appears weak. The distribution of disease both in terms of its prevalence and its severity could help argue the case for their usage. At best it would appear that a targeted subgroup might benefit from the application on sealants on selected teeth. Should specific patterns in the distribution of dental caries exist the case for adoption of sealants would be stronger.

2.2.3: Strategy Approach

Rose (1993) divides strategy approaches into two distinct groups: those aimed at the population and those in which certain sections of the population are identified, either as a group or as individuals, the risk approach. To distinguish between the two aspects of the risk approach, that in which groups are identified is termed a directed population approach, and that in which individuals are identified, is

termed a high risk approach. Rose poses the fundamental question: does a small increase in risk in a large number of individuals generate more cases than a large increase in risk in a few individuals?

In general medical care options for varying strategy approaches have been suggested. Ritson (1994) examined differing strategies for alcohol-related problems, concluding that no single approach worked. This conclusion was also drawn by Rose (1993) and highlighted that no single approach should be adopted. Differing strategic approaches have been reported for coronary heart disease (Kottke et al., 1985; Lewis, 1988; McMichael, 1989) and hypertension (Watt, 1989). A common finding in all the papers was that an approach based only on a high risk approach was insufficient to prevent the problem, indeed (Kottke et al., 1985) concluded that '...only a population approach can prevent the majority of deaths from cardiovascular disease in a community.'.

The changes in the distribution of dental caries described previously have lead to certain authors to state that the majority of caries is to be found in the minority of children, (Pitts et al., 1994; Pitts and Palmer, 1995; Pitts and Evans, 1996; Pitts et al., 1997). Pitts (1997) when posing the question as to whether the profession understood which children need care and get appropriate care, concluded that '...more integrated approaches to prevention should be focused on identifying those children who still have high levels of dental need..'. This approach is synonymous with risk measurement: the identification of individuals with a greater risk of developing future disease when compared to others: the risk approach. Pitts (1997) made the distinction between low risk and high risk using a dmf score of 2. The basis for this cut-off point is not provided. Furthermore the assumption is being made that those individuals who currently have the highest levels of caries will continue to develop the highest future increments. Both Johnson (1991), based on the proceedings of a symposium in London, and the

more recent symposia held in North Carolina (Newbrun and Leverett, 1990) suggest that this pattern of increment development may not be the case. Both came to similar conclusions regarding caries markers, namely that no single marker is ever likely to be satisfactory. This view has been reinforced by Hausen et al. (1994), who highlighted the limitations of current screening mechanisms, without which the efficiency arguments for adopting a risk approach remain lacking, particularly that focusing on individuals.

The risk approach in dentistry has also be challenged by Sheiham (1996). He argues that the recent decline in both caries and periodontal diseases has occurred through an uncoordinated adoption of the population approach. Furthermore, he highlights similar limitations in adopting the high-risk approach to those reported by authors examining general chronic diseases. First, as Rose (1993) also comments, there is the implication that the problem belongs to someone else, the 'high-risk' group. The majority of the population are considered to be 'safe'. Second, the approach is both palliative and temporary in nature: it neither addresses the underlying cause of the problem nor prevents new cases occurring. Finally, to be successful, the high-risk approach requires individuals to adopt differing social norms compared to their peers.

In summary, three approaches exist: the population approach, in which no attempt is made to identify those groups of individuals which receive an intervention; the directed population approach, in which, although some selectivity in resource allocation is made it is not at an individual level; and the high risk approach, in which individuals are screened and selected for the intervention.

The theoretical basis for a risk approach centres on targeting limited resources to those in greatest need, implicit is both an index of risk and a mechanism to apply it. Whether practically this approach is more efficient will depend upon both the

cost and accuracy of the mechanisms used to identify those at greater risk and the effectiveness of the targeted interventions.

For general chronic disease problems the consensus appears to be that a strategy based on a population approach is far more likely succeed than a risk based approach. In dentistry, while there remains a theoretical basis for the risk approach, data are lacking to justify its adoption. Indeed, a critical assessment of the application of strategic approaches, in terms of both content and cost, has yet to be undertaken.

2.3: Health Economic Aspects of Strategy Selection

Despite the fall in dental caries over the last decades the costs of dental health care have continued to rise (OECD, 1990). As with health care in general, this has been above the general rate of inflation (Schieber, 1987). While this rise is due to a large number of factors, such as the price of materials, personnel wages, use of more advanced technology, a more litigious consumer, or, even partly, an increase in growth of the number of consumers, there is little evidence that the increased spending has contributed to better health (Abel-Smith, 1996). Increased spending however may not necessarily be inappropriate. If better dental health is a result of this then a society may well accept the rise in costs. The enlarging concern related to health care spending suggests that societies are becoming wary. No longer are the medical professions seen as being above reproach. Consumerism has started to penetrate the health sectors and with it, the need to justify the use of resources. This has led to the handling of health care, at least partially, as an economic commodity (Kobelt, 1996). Fundamental to this argument is the precept of expected utility theory (Torrance, 1987). This theory postulates that a person, in this case a consumer of oral health care, is faced with

uncertainty but free to make a decision. This argument requires a number of prerequisites. First, the consumer of oral health care has the knowledge to select all possibilities of goods according to his or her preferences. Secondly, when the consumer is given a number of options, and the first choice is preferred to the second, and the second is preferred to the third, then the first is preferred to the third. Finally the consumer will always aim for the most preferred state. However there are problems with these assumptions.

First, whilst the profession's knowledge may be incomplete it is likely to be more complete than that of the consumer. Secondly, if all options are to be considered, the options to spend resources on preventing a tooth becoming carious or treating it, the longevity of the restoration following treatment would need to be known. The data necessary to answer this are not available. With this lack of knowledge how can an ideal decision be made?

2.3.1: Possible methodologies

There are many methods available for the economic evaluation of health programmes. According to Drummond et al. (1987) the types of evaluation can be classified into a four celled matrix, Figure 2. The two axis are questions that the planner needs to answer;

(i) is there a comparison of two or more alternatives, and

(ii) are both the costs (inputs) and consequences (outputs) of the alternatives examined?

Three of the cells result in only a partial evaluation of a programme. While the information available may only allow planners to do such studies, the usefulness

Figure 2: Types of economic evaluation

Are both costs (inputs) and consequences (outputs) of the alternatives examined?

Is there comparison of two or more alternatives?	**NO**	**examines only consequences**	**examines only costs**	**PARTIAL EVALUATION**
		PARTIAL EVALUATION		
	NO	outcome description	cost description	cost outcome description
		PARTIAL EVALUATION		**FULL EVALUATION**
	YES	efficacy or effectiveness evaluation	cost analysis	cost-minimisation.cost-effectiveness, cost utility, and cost-benefit studies

of such analyses may be questioned. The fourth cell, formed by knowing both the inputs and outputs and making a comparison between two or more alternative programmes gives rise to four possible evaluation analyses;

cost-minimisation
cost-effectiveness,
cost-utility, and
cost-benefit.

In order to apply any of the above types of analyses the components of the evaluation need to be identified. The inputs, the resources consumed, need to be measured. These are called the costs and can be subdivided into three; direct costs, indirect costs and intangible costs. The direct costs are largely in the health care sector. They include wages and consumables. When considering a fluoride rinse programme these would be the cost of the rinse used and health care personnel involved. However the costs may be incurred in other sectors. Teachers paid through the education budget may be used to implement the fluoride rinse programme instead of health care personnel. Here the costs would come from the education budget.

Indirect costs, sometimes called production losses, occur when an individual is unable to work and produce whilst receiving therapy. Professionally applied fluoride gels requiring an individual to attend a dental surgery, would have higher indirect costs compared to water fluoridation requiring no loss in production time. This raises a major issue in economic evaluation. If production losses are going to be a major factor then those groups not producing are at a advantage. When faced with only an economic choice, a programme which is formulated for the 'non-producing' groups would be best when compared to a programme targeted at a 'producing' group when the same outputs exist. On costings, a programme

particularly values children, those outside the recognised workforce, for example housewives, and those retired. The opposite is true when examining benefits with those in work being valued more.

Intangible costs include pain and suffering. These are difficult to measure not only qualitatively but quantitatively. In oral diseases, where the majority of disease processes are chronic in nature, these costs may be large.

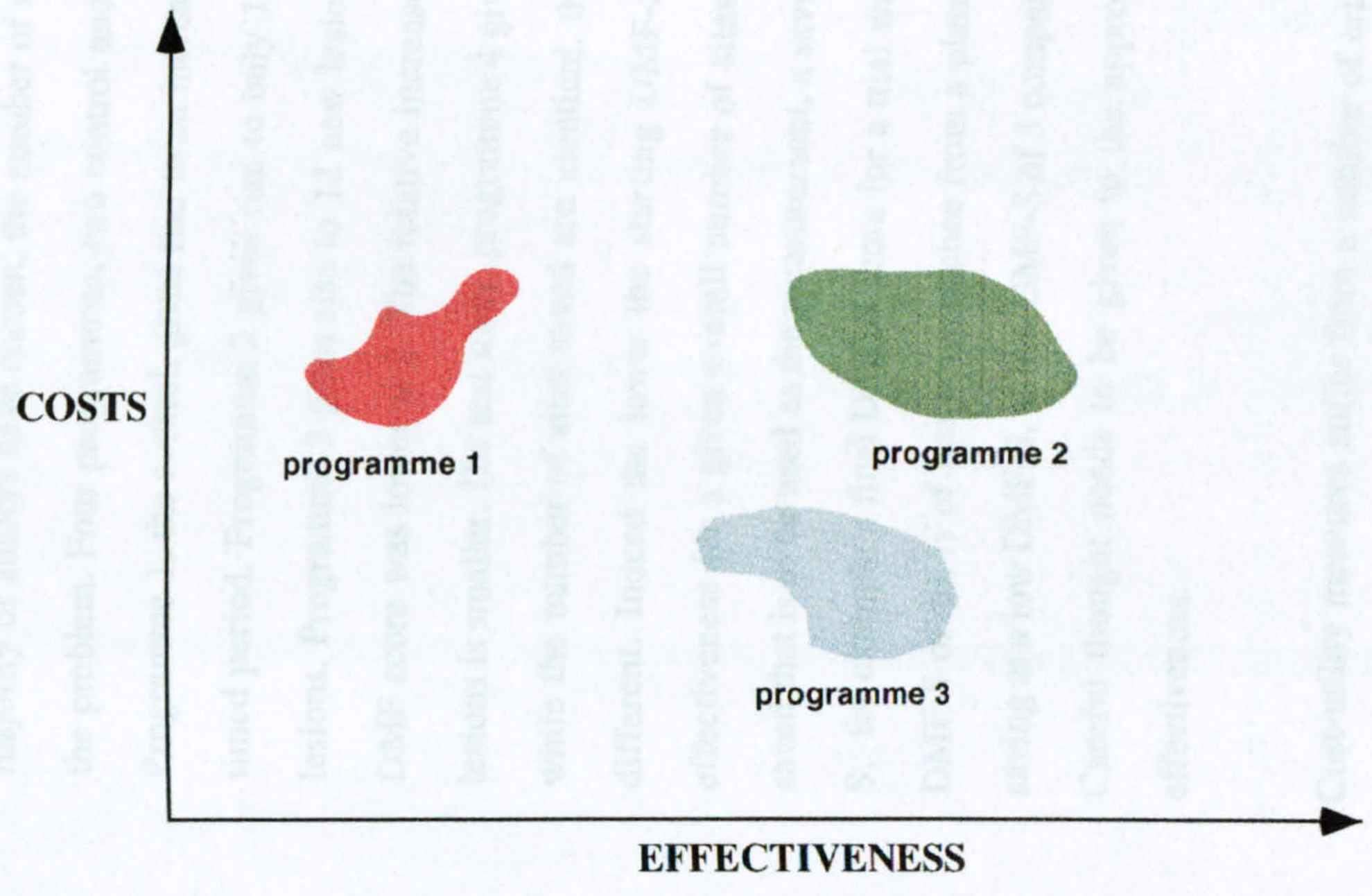
The outputs, namely improvements in health, also need measurement. The measurements adopted will depend upon the type of evaluation adopted. For cost-effectiveness studies natural units are used. For dental caries the number of tooth sites saved would be an appropriate measurement. For cost-utility studies utility measurements are adopted. The most frequently adopted unit is that of quality adjusted life years, QALYs. These are arrived at using life-years gained adjusted by a series of utility weights reflecting the relative values individuals place on different states of health, (Kobelt, 1996). For example a caries free dentition might have great importance for a young individual, but this might not be valued so highly in later life. Finally for cost-benefit analyses the choices are presented in monetary units. The measurements are a summation of three units: the direct benefits, for example, savings in dental treatment; indirect benefits, for example, an earlier return to production; and the intangible benefits such as an individual feeling healthier.

Cost-minimisation analyses are a specific form of cost-effectiveness studies. The outcomes of the programmes being compared are tested through controlled clinical trials, ideally running concurrently. If the outcomes of the programmes are the same in clinical terms then one need only compare the costs of each programme, the most appropriate being that in which the costs are the least.

However, such analyses of programmes are rare. It requires that the outcomes of two or more programmes are identical.

However, comparison can be made without valuing the consequences. The objective of the planner is to identify those variables that will maximise the value of the system subject to an assumed set of conditions. One example is the preservation of a tooth. If this is regarded as of overriding importance, the costs of keeping the tooth utilising differing approaches are compared. While alternative treatments may vary in the number of years that a tooth is saved, a comparison can be made in cost per year of a tooth saved. This would provide information as to how the resources allocated to saving teeth should be spent to maximise the total number of added years of tooth life. It should be noted that the costs are not necessarily kept to a minimum. Packer (1968), introduces a further feature that should be included when making an analysis, uncertainty. The uncertainty results from the inability to predict the variable and parameter values for the time period relevant to the alternative programmes being considered. Prediction difficulties arise from two sources. First many relations are not fully understood, and second, it is impossible to know whether the relationship that exists between variables currently will continue to do so. In dental caries an example of the first type of uncertainty is the relationship between sugar, fluoride and dental caries. An example of the second type of uncertainty is the possibility of a vaccine being discovered which completely invalidates current concepts of preventative dental care. In Figure 3 an example of three programmes is illustrated. For each programme an area of uncertainty is allowed for. For programme 1 the costs are equal to those of programme 2 under all circumstances. In addition, the effectiveness of programme 2 is greater under all circumstances. Thus programme 1 can be disregarded. In programme 2, while the costs are greater than programme 3 under all circumstances, its effectiveness in certain circumstances is greater. The choice is then left to the policy maker as to which programme is the

Figure 3: Cost effectiveness distributions for three programmes



more appropriate. The more accurate and appropriate the information, the smaller the areas of uncertainty, the easier the choice of strategy.

While costs may be quantified with reasonable ease, effectiveness is more difficult. In caries prevention is it the change in the percentage of subjects in a population who are caries free, is it the percentage reduction or, as the vast majority of authors have chosen, the number of sites saved? Figure 4 illustrates the problem. Four programmes, two control and two test studies are analysed. Programme 1, the control, gives rise to an increase of 20 carious lesions over a timed period. Programme 2 gives rise to only 10 new lesions, a saving of 10 lesions. Programme 3 gives rise to 12 new lesions. However the starting total DMF score was lower at 2. The relative increase for the second study in new lesions is smaller. The test study, programme 4 gives rise to 2 new lesions. Thus, while the number of sites saved are identical, the percentage effectiveness is different. Indeed the lower the starting DMF-S the greater the percentage effectiveness for a given overall number of sites saved. However if it is sites saved that is to be used as the measurement, a saving of one site at a high DMF-S, for example a final DMF-S score for a trial was 39 compared to the control DMF-S of 40, is of less consequence from a planners viewpoint than the same saving at a low DMF-S, a final DMF-S of 3 compared to the control DMF-S of 4. Careful thought needs to be given to the appropriate method of measuring effectiveness.

Cost-utility measures suffer from a number of criticisms largely related to the measurement of health. McGuire et al. (1988) highlight a number of contentious areas. First, a person's response to a hypothetical situation may well be different to a real situation. However for a preventative programme the planner needs to make the choice before an ill-health state occurs. Second, most of the measurements that have been undertaken reflect a move from a position of good

Figure 4: Effectiveness of two caries preventive programmes

	Programme 1: control group	Programme 2	Programme 3: control group	Programme 4
Initial DMF-S	5	5	2	2
Final DMF-S	25	15	14	4
Sites saved		10		10
Percentage effectiveness		50		83

health to worse health states. They do not reflect the value of moving from a chronic health state to good health or from one condition to another. If health is subject to the law of diminishing marginal utility then this may be an important source of error if the results from the former situations were applied to the later situations. Third, the valuations elicited will be affected by the expected duration of the condition. Suffering from a chronic condition for two years may be more than twice as bad as suffering for one year. Fourth, valuations given may change with the way in which the question is asked. Torrance (1987) developed two techniques to elicit valuations of health state, time trade off or the standard gamble technique.

Time trade off is a option in which patients are asked to make a trade-off between a chronic health condition, y, and good health for a shorter period of time, x. The period x is altered until the individual is neutral in choice selection. The valuation, h, of the time trade off is x/y. In the standard gamble an individual is asked to make a trade-off between the certainty of having for z years the chronic health condition, or make a gamble between good health for the z years and death. The probabilities of the gamble between health and death are then altered until the individual is indifferent between the gamble and the chronic health condition with certainty. At the point of indifference the valuation of the health condition is calculated as being the same as the probability of health in the gamble.

Finally cost benefit analyses conceptually take the decision making process a stage further, and as such, suffer from many of the criticisms raised for cost-utility measurements. The assessment of a programme, by having converted all costs and benefits to a common denominator, is simply: do the benefits exceed the costs? This allows both similar and widely differing programmes to be compared. In theory cost-benefit assessment is the ideal. However there are number of major flaws. First, when choosing a programme financial constraints are ignored. The

argument as to whether, having selected the programme using cost-benefit analysis techniques, it is to be adopted is dependant upon whether funds are available. Second, and in common with cost utility, the measurement of disease states before they occur is problematic. Furthermore there is no one accepted procedure in dealing with the time component. Prevention is very much a benefit for the future, yet the value that an individual, and hence the level of benefit, places on a dental caries free dentition may alter. Consider the following example. An individual has a sound dentition which will, at a later date, require orthodontic extractions. If a preventive measure is applied now, the teeth that are to be extracted will remain caries free. If no preventive measure was taken the teeth would suffer a dental caries attack, but not severe enough to cause any discomfort. In this scenario the cost-benefit analysis would suggest that the use of the preventative measure was invalid. If however, the child was to suffer discomfort, the cost could change the outcome of the analysis.

A further issue relates to the guiding principles of the preventative programme. In public health there may well be a conflict between the costs and benefits. Those who pay more may well benefit the least. In private business cost-benefit studies would appear to be more appropriate. Both the costs and the benefits accrue to the same body. For public health there is likely to be a conflict between the efficient use of resources, giving the lowest cost:benefit ratio, and equity.

In conclusion it would appear that, while health economics can aid in the decision making process, the majority of current methodologies have serious shortfalls. These shortfalls largely concern the value that populations put on a sound dentition and how that value changes with time. When choosing between the various methodologies the arguments would favour cost-effectiveness studies. They have at least set a goal for policy makers to reach. While the costs can be estimated in reaching these goals, it is the measurement of effectiveness that needs

to be formulated. This requires knowledge of a valid scientific basis for the prevention of dental caries.

2.3.2: Health economics as applied to dental caries prevention

Foch (1981) reviewed the application of health economics to preventive dental care programmes. One of his main conclusions was the lack of 'real' studies. With a few exceptions the studies reported on were theoretical in nature, and the results dependant upon a number of suppositions which were open to question. The studies included assessments of both single agent measures, such as fluoride, and multiple agents, for example combining fluoride with fissure sealant applications.

In a series of articles Davies (1973a; 1973b; 1973c; 1973d; 1973e; 1973f) applied the principles of cost-benefit analyses to differing fluoride agents. However the methodology applied used a simplistic formula to calculate the cost-benefit ratio. The ratio was derived by dividing the cost of implementing the programme by the cost of treatment that would have occurred had the programme not taken place. This methodology ignores both the indirect and intangible costs and benefits. Furthermore Davies assumed a one off action. This ignores at least two facts. First, caries is a lifelong process. As such it is the continued exposure to a preventative agent that is required. Second the half-life of a restoration is related to the size of the restoration (Elderton, 1993). Each replacement restoration is likely to be bigger, and in consequence, both more expensive and have a shorter half-life. Furthermore it is assumed that there is a unmet demand for dental care. By not having to replace a restoration the operator can offer benefits through other aspects of dental care. Work by Birch (1988) in the United Kingdom and Grytten

(1991) in Scandinavia suggest that there would appear to be lower aggregate cost savings from fluoridation than expected.

Klock (1980) used both cost-benefit and cost-effective methodologies to assess a preventive programme that included fluorides and fissure sealants. He found that, in spite of a marked reduction in caries activity, both cost benefit analysis and cost effectiveness analysis indicated that the preventive programme was highly uneconomic compared to traditional dental care. Further examples of the application of cost benefit methodology are Grainger (1973), who applied it to dental services in general, and Geiser and Menz (1976) to public dental programmes. The latter made the relevant point that the summation of benefits accrue over a long period of time.

The majority of cost benefit studies have measured the benefits of fluoridation by either using indices of dental caries severity to determine the reduction in the need for dental services or by directly measuring the change in dental services attributable to fluoridation. These benefits are then priced and compared with the costs of fluoridation. Nearly all the studies ignore the time component. Nelson and Swint (1976) applied the methodology prospectively to the fluoridation of an unfluoridated town. They demonstrated, hypothetically, how the time factor could be handled in an analysis, suggesting that it has considerable implications for the end result.

O'Rouke et al. (1988) examined both the costs and benefits of running a 3 year fluoride tablet programme in Manchester. The tangible benefits were calculated using a resource related index, and for the permanent teeth a saving of £1.87 was given. The cost of the tablet programme was calculated at £4.39. However when the intangible benefits were added to the equation, such as the reduction in pain and provision of anaesthesia, the equation appeared more favourable.

Nevertheless it was concluded that to obtain a favourable cost: benefit ratio there must be an adequate level of dental caries activity.

The application of cost-effectiveness methodology to dental caries preventive programmes is more extensive. However, as with cost-benefit methodologies, these are in the majority hypothetical situations. Indeed a review by Boggs (1973) highlights "... limitations in lack of scientific rigour, in lack of adequate evaluation, and the presence of considerable guessing,..". Studies carried out include those involving the use of single fluoride agents, as by Birch (1990), combinations of fluoride agents, for example Bohannan et al. (1985) and fissure sealants, Mitchell and Murray (1989).

Dowell (1976) examined the economics of water fluoridation and concluded that the cost per unit benefit was reduced as the size of the population increased. Birch (1990) suggested that cost-effectiveness estimates should be assessed not only upon the severity of dental caries but, as with Dowell, also the size of the population. As he points out, the cost per unit benefit associated with fluoridating a small population of high dental caries levels is roughly the same as for fluoridating a population ten times as large but with low caries levels. In a concluding statement of a workshop examining the cost effectiveness of caries prevention in dental public health, Burt (1984) stated "..it (the workshop) left little doubt that water fluoridation does indeed result in cost savings, one of the very few public health actions to do so." However, even this statement was qualified by the rider that the extent of these savings was unknown and required further research. Possible savings ranged from 30 cents to $12 per DMF surface saved, a 40 fold variation. The Federation Dentaire Internationale (F.D.I.) (1981; 1984) had come to similar conclusions as that of Burt regarding water fluoridation.

Heifetz et al.(1992) reported the findings of various topical fluoride agents on hypothetical populations with various estimates concerning the dental caries increment and effectiveness of the agents, the data being presented as the cost per surface saved. Whilst the assumptions made in the calculation are open to question, the study did highlight the enormous range in the cost-effectiveness of agents. They ranged from $1.00 for weekly mouth rinsing using 0.2% NaF solution to $21.30 for daily APF gel (0.5% F) in customised trays.

In a review of the cost-effectiveness of school-based preventive dental care, Klein et al. (1985) found ".. that dental health lessons, brushing and flossing, fluoride tablets and mouth rinsing, and professionally applied topical fluorides were not effective in reducing a substantial amount of dental decay, even when all of these procedures were used together.". The findings also reinforced previous findings that water fluoridation was the most cost-effective method of reducing dental caries.

Doherty (1990) examining school based mouth rinsing programmes found that as the programme size increased the returns decreased. This could be due to organisational constraints, in terms of limitations of efficient management. Problems arose in those preventive programmes which were labour intensive such as school based individual measures, as opposed to water fluoridation, which are more of a public health measure. Indeed in one study, despite the problems in co-ordinating and finding volunteers for a large programme, they were more productive, and in consequence, more cost effective, than paid workers (Doherty and Martie, 1988).

In reviewing the evidence on cost-effectiveness of fissure sealants Mitchell and Murray (1989) highlighted the paucity of literature on the subject. Furthermore they raised the issue about the changing effectiveness of the materials. It was

reported that the results of more studies, particularly those which evaluated fissure sealants as an integral part of routine dental care, were promising. However factors such as the durability of the material and tooth selection greatly influence the viability of fissure sealants. In particular they agreed with the findings of Eklund and Ismail (1986) that, as the dental caries prevalence declines, the economic returns are smaller. Indeed, given that lesion size, and in consequence the restorative procedure required, will also be smaller as the prevalence declines fissure sealants may no longer be either cheaper or as effective as conventional restorations. Pavi et al. (1992) came to these conclusions when examining the costs and effects of preventive dentistry in a Glaswegian health centre. The costs and effects for 7 year-old children were measured over a 4 year period. It was concluded that the preventative package was more costly than restorative care due to the low dental caries increments. Further reviews of the cost effectiveness of fissure sealants include the work by Horowitz (1980) examining their role in private practice, and Burt (1985b). The former study concluded that, although estimates of the costs of the use of sealants were high when compared to other preventive measures, they still had a considerable value. This finding was based at disease levels in which 50% of lesions were to be found on surfaces where sealants could act. In the latter study particular emphasis was placed on the wide range of costs of placing fissure sealants, a similar finding to that by Brunelle (1976).

2.3.3: Summary of health economic aspects of strategy selection

In conclusion, current health economic methodologies have major shortcomings when applied to dental caries prevention. Although not unique when compared to general health analyses, the two main flaws centre around measurement of a chronic disease and the assessment of effectiveness. Of the current methodologies

available, cost-effectiveness is the most appropriate. Cost-benefit and cost-utility both require a satisfactory index to assess intangible benefits such as freedom from toothache, possible fear of dentists, and freedom from requiring anaesthesia. By using cost-effectiveness studies an appropriate goal can be set by the planner and the differing programmes compared. There are problems relating to cost and effectiveness measurement and no single methodology yet provides a totally adequate solution to the problem of economic assessment of caries prevention. However some common themes arise from the review. First, the type and scale of the programme is important. The more individualised the programme, the more labour intensive, and in consequence the more costly. Secondly disease levels are crucial. The higher the levels of caries at the commencement of a programme, the greater the scope for a reduction , and in turn, the more cost-effective the programme is likely to be. Thus at high dental caries levels the majority of preventive programmes are a better solution than a treatment regime. At low levels of disease this may not be the case.

Combinations of fluoride agents give conflicting results. The majority indicate that, after the first agent, additional programmes do not yield a substantial economic return. There are little data concerning the economics of fissure sealants. However, as with fluorides, the distribution pattern of disease both within the mouth and within the population has large implications. In general, the techniques for both analysing costs and the measurement of efficiency vary considerably. A major advance would be a more scientific formulation to estimate the efficiency and effectiveness of agents when planning dental caries preventive strategies. Quantitative data on the impact of preventive regimes in the number of tooth sites saved per unit cost would allow a scientific assessment to be made. The formulation requires an assessment of the epidemiology of caries: what is the impact in change in caries levels mean in terms of the numbers and distribution of teeth and sites affected.

2.4: Summary

A number of conclusions can be drawn from the review. First, there would appear to be patterns in both the presentation and development of dental caries. Dental caries presents symmetrically: the evidence suggest that in addition to homologous paired symmetry, other relationships exist. Certain groupings of teeth have similar susceptibilities. The most susceptible tooth would appear to be in the first molar. This in part may be due to the statistical methodology employed when analysing dental caries data.

Second, in addition to the susceptibility of the various teeth there is a pattern of tooth surface susceptibility, the occlusal surface, if present, being the most susceptible. It would also appear that the pattern of dental caries attack is constant unless the force attacking the site is of a very low level and the morphology of that surface is modified, due to attrition or abrasion or even dental care intervention. In addition what data are available would suggest that fluoride, which reduces the attack rate, does not modify this pattern. This suggests that fluoride does not have a greater effect on smooth or approximal surfaces when compared to pit and fissure surfaces. What has been observed is a reduction in the attacking force which, in consequence, has been insufficient to overcome the resistance factor of those, the least vulnerable, sites.

These conclusions highlight a number of very important issues when proposing the development of caries preventive regimes. First, there is an order to the development of dental caries: certain sites and teeth are more prone to caries than others. At low DMF scores it is the pit and fissured surfaces of molar teeth that will develop caries. Secondly, as the attacking force decreases, the effect may not present at a constant rate. The work concerning the pattern of attack in the anterior teeth would suggest that a number of sites could have similar vulnerability levels.

Thus a decline at high levels of caries would be far easier to achieve than at lower levels of caries. To obtain a reduction of 5 at a DMF-S of 20 requires less effort than at a DMF-S of 6. Thirdly, the impact of fluoride would seem to affect the overall attack rate, and not impact on certain sites more than others. All these issues will influence the selection of preventive agents to be used in a scientific approach to the prevention of dental caries.

Finally, while the above findings deal with aspects of the scientific formulation of a preventive strategy other aspects should be considered. In particular, the review of the application of economic methodologies highlight the shortcomings. Although this project will deal with the identification of the scientific basis, the justification for adoption will require further work.

3: METHODOLOGY

3.1: Introduction.

This section outlines the basis for the methodology adopted for testing a conceptual model for the epidemiological characteristics of dental caries relevant to planning preventive strategies. The literature review on the epidemiology of dental caries suggests certain features in the 'at population' level and at the level of mouth and tooth, the latter will be called the 'within mouth' level.

At the population level, the distribution of caries would appear to be related to the severity of the attack. In populations with high caries levels, the distribution appears more widespread than in populations with low caries levels: changes in levels appear to affect the whole population and not selected groups. At the within mouth level, not all teeth or tooth sites appear to be equally prone to caries. Certain teeth or tooth sites appear more likely to become carious than others. To test these concepts a theoretical model is described and tested. The model proposed suggests that the distribution of dental caries at both the population and at the within mouth level have set parameters.

Caries data measurement is discrete: no matter how accurate the diagnosis of whether a site or tooth has undergone a caries attack, the answer will remain dichotomous and cannot be refined. This can create problems when examining the distributive properties of caries within a population. At the two extremes of possible caries distributions, at DMF-T scores approaching 0 and 32 and DMF-S scores of 0 and 128, the distributions will become skewed. This will affect any relationship between the mean and the variance. For any distribution which is tending toward the extremes, the variance will reduce the closer the mean approaches the extremes. However this does not detract from the usefulness of

the knowledge of the relationship. Even accepting that the relationship may, in part, be determined at the extremes, the actual mathematical value describing it, should it exist, can be used to ascertain the proportion of the population at each of the discreet intervals. In addition, the fact that the data are discreet does not necessarily imply that a relationship between the mean and variance exists.

These parameters include four relationships. First, the relationship between prevalence and the mean DMF score. For a given prevalence, it is hypothesised that the mean is constant. Second, the relationship between the mean DMF and the variance. As the mean DMF score changes, it is hypothesised that there is a change in the variance, and that the change in the variance is a function of the mean. Third, for a given prevalence, it is proposed that the distribution within a population is relatively constant, and, should the first and second relationships hold, this will also be true for a given mean DMF. For a given prevalence or mean DMF there is a constant percentage of a population at each DMF score. Fourth, at the within mouth level, for a given DMF score, it is hypothesised that there is a hierarchy of tooth attack. The conceptual model to be tested proposes that, for a population in which either the prevalence of mean DMF is known, the distribution of lesions will be constant.

Although two of the hypotheses outlined in the model may apply to any distribution of discreet data, the actual values of either of the relationships, that between the mean and percentage caries free and that between the mean and the variance, would be of critical importance in developing a scientific basis for caries preventive strategies in establishing the impact of regimes in terms of the percentage change at each interval.

The hypotheses necessary to verify the conceptual model will be developed in two stages. The first stage will identify the relationships outlined above; between the prevalence and mean score and those of the distributive aspects of caries at both a population and within mouth level. In the second stage the model identified in the initial stage will be tested using differing data sets.

3.2: Issues in data analyses.

Currently any assessment of the effectiveness of preventive programmes uses the DMF index, (Klein et al., 1938). For example in a clinical trial, a difference in DMF scores between groups suggests a level of effect. Mean DMF-S or DMF-T scores refer to the severity of the disease: the effectiveness of a caries preventive programme is the change in the severity of the disease. A decrease in a trial group when compared to a control group for a preventive strategy would suggest that a preventive programme has been effective.

However there are a number of issues when ascribing changes in the DMF score to caries, particularly when using cohort or longitudinal trial data. These issues are highlighted by Drake et al. (1997). They include changes in the DMF score for reasons other than caries attack: the criteria used by practitioners when treating those individuals involved in the study during the duration of the trial may be different from the epidemiological criteria for intervention. Beck and Drake (1997) cite examples from the Piedmont 65+ Dental Study. First, the issue of whether the F component of the DMF index is true caries experience. Differences were found in the degree of association between root and coronal caries when using incidence rates of new caries experience and untreated decay and ethnic background. Those individuals of white origin were found to have higher levels of treated caries than individuals of black origin. The interpretation of the results

by the authors was that fillings, placed with unknown diagnostic criteria, attenuated the relationship.

The second issue relates to the number of teeth or sites that are present in the mouth. Only sites that have erupted can become carious. During the study period teeth may erupt or be lost. This could impact on analyses in a number of ways. First, if not all sites have erupted, establishing whether all sites in a full dentition are equally prone to caries attack would not be possible. For example, at 8 years of age, the first molars are likely to have erupted, but not the second molars. In consequence it would be impossible to establish whether the first molar tooth was more prone to caries than the second.

Secondly, but allied to the first issue, as a tooth erupts the sites may have differing levels of resistance. Unless the resistance for each of the sites on newly erupted teeth is identical to those teeth already in the mouth then the increase in the number of teeth during a trial will again prevent an accurate assessment of the effectiveness of caries preventive agents. In addition, the resistance for each site should remain constant during the trial period, but this may not be the case. The literature reviewed suggests that, following eruption, enamel undergoes a period of maturation. Brudevold et al. (1982) argue that during this period of maturation the resistance changes and increases. For a given attack rate, the outcomes would be different in newly erupted and 'mature' teeth. Teeth newly erupted would cavitate earlier than teeth with mature enamel. Current methodology fails to take this into account. The adoption of caries increments to assess the effectiveness of caries preventive agents during periods of tooth eruption and up to 5 years post-eruption must take the changes in enamel resistance factors into account.

A further flaw in current methodology using DMF relates to the fact that the DMF index is non-reversible. Being non-reversible the programme that is being

assessed is not detecting whether the attack rate has been reduced to a level at which those sites that have already cavitated would not do so. If a decrease in the attack rate has occurred, it would be impossible to detect that change using the DMF index. In those cases where cavitation has not occurred, the process was not of sufficient strength to cause cavitation within a given time period.

Consequently to assess accurately any preventive programme there needs to be an increase in DMF in both the trial and control groups. One cannot determine whether the process, and consequently the attack rate, has changed except that it has increased. When formulating caries preventive strategies it becomes difficult to ascertain to what extent the methods adopted are working. No change in the DMF score does not imply that the attack rate has decreased. The host resistance factors may have increased relative to the attacking force. Despite an increase in the attacking force, that increase may still be insufficient to overcome the resistance. Furthermore an increase in the DMF score may not necessarily indicate an increase in attack intensity. As discussed previously, any newly erupted sites may have a lower resistance than those sites already present in the mouth. If certain sites have a differing resistance, those sites at which the potential has not been overcome will not decay until the attack rate increases.

This creates a problem when analysing cavitation changes over time. For example, suppose at seven years of age an individual has a DMF of 4. This is the result of a given attack intensity. The attack intensity is sufficient to only cause cavitation on the occlusal surfaces of the first molars. By 12 years of age the same individual has had an increase in the number of teeth in the mouth. Those newly erupted teeth will have a range of resistance less than, the same or greater than those of the first molar teeth. If the attack rate has remained constant, and the site resistance factors are the same or less than those of the occlusal surfaces of the first molar teeth, the sites will cavitate. If the attack rate has been reduced, but the

site resistance factor is lower than that of the occlusal first molar surfaces, the newly exposed surfaces may cavitate. Thus, despite a reduction in the attack rate which would have resulted in no cavitation in the first molar teeth, the programme would show an increase in the DMF score.

3.3: Data analysis methodology.

The statistical methodology used to develop the conceptual model varies according to each of the four relationships outlined in section 3.1. The first, that of the relationship between the mean DMF score and the prevalence uses correlation methodology. Conventional correlation coefficients are measures of the linear relationship between two variables, and in consequence will underestimate those variables having a non-linear association. An index which does not assume a linear relationship is the correlation ratio, and this measure should be utilised whenever a scattergram of the data suggests a non-linear relationship exists, (Norusis, 1990). This methodology is also used to examine the relationship between the mean and variance. The third relationship, that of the variation in distribution of each DMF score according to the mean score uses simple descriptive statistics.

For the fourth relationship, the 'within mouth' relationship, the data are analysed using probit analysis, a methodology for examining dose-response relationships where the dependent variable, in this case caries, is dichotomous, that is whether the site had experienced caries or not. Cavitation is measured as qualitative data. Caries rates are only recorded as a change from one state to another, not on a continuum. The dependant variable, DMF, is dichotomous. The question is: what dose of force is required to cause cavitation when the effect is measured it is either an all-or-none response? The tooth site is either cavitated or it is not. To overcome

the problems highlighted in Section 3.2, the actual proportion of each tooth site recorded carious at each DMF-S was calculated and replaced with the value of the standard normal curve below which the observed proportion of the area is found. Obviously, when using standard normal values, negative scores can occur. To overcome this, the constant 5 was added. For example, at a DMF-S of 1 for a particular site if half of the subjects studied had caries, the probit value would be 0, since half of the area in a standard normal falls below a z score of 0. When the constant is added, the transformed value for the proportion becomes 5. If the observed proportion of individuals in whom the site was carious was 0.84, the probit value would be .3413, i.e. a z score of 1, which would give a transformed value of 6.

Since all tooth surfaces do not necessarily respond in a similar manner, the problem must be formulated in terms of the proportion responding at each level of the stimulus. To estimate the effects of one or more independent variables on the dependant variable probit analysis should be used. When using probit analysis, any changes are constant in proportion, and thus changes on a log scale are then constant. In a probit transformation each of the observed proportions are replaced with the value of the standard normal curve below which the observed proportion of the area is found. For example, if half the subjects in a caries trial had one particular site carious, the probit value would be 0, since half the area in a standard normal distribution falls below a z score of 0.

Consider a change in DMF from 10 to 15. In absolute terms it is an increase in DMF of 5, that is a 50% increase which has a natural logarithmic value of 0.405. A change in DMF from 6 to 9 in absolute terms whilst only an increase of 3, is also a 50% increase, that is the same natural logarithmic value. This logarithmic transformation of the data provides a linear relationship between the probability of an event occurring for any given value. The values can be calculated by plotting,

for a given probability for each tooth site, the numerical value of the DMF-S at which this would cavitation occur. The ratio of the DMF-S values for each tooth surface will be equivalent to the relative weighting of the resistance factors. When these weightings are applied, the effectiveness of a preventive programme should be able to be assessed accurately.

To establish whether a hierarchy exists, the probability of finding each site carious for a given DMF-S score was established. The probability was calculated by adopting a common reference value for the proportion of the tooth sites which become carious; in this project 0.5 was used, although any value of proportion can be used. The question can then be phrased in terms of the value of the DMF-S at which 50% of the sites or teeth would expected to have cavitated. The probability scores derived are then aggregated to produce an overall picture of tooth and site susceptibilities.

The data on each tooth surface was converted to a dichotomous state: 0, no evidence of caries attack, and 1, caries attack had taken place, that is the surface was decayed, missing or filled. For each DMF-S score, the percentage of tooth surfaces that had undergone a carious attack was calculated, to give a probability score of between 0 and 1. Subsequently, the log transformation of the probabilities for each DMF-S against the actual DMF-S score was plotted for every surface. The common reference value, outlined above, 0.5, was used to establish the susceptibility of each surface to a given caries challenge. As some random variation can be expected, the susceptibilities are grouped within bands rather than treated as individual sites.

3.4: The data sets.

This project uses data obtained from three sources: the National Preventive Dentistry Demonstration Programme (NPDDP) in the United States; data from the annual surveys of 12 and 14-year old children carried out in the United Kingdom organised by the British Association for the Study of Community Dentistry (BASCD); and a 4 year longitudinal study carried out by the School of Medicine and Dentistry, University of Wales and Walsall Health Authority.

The NPDDP data set was used as it contains the most extensive and comprehensive longitudinal data available on caries preventive regimes using standardised criteria. which have been shown to be reliable through extensive critical analyses of the project. Perhaps most importantly for this project, the caries data range was very wide: both within the individual ethnic groups and according to fluoride status. The NPDDP project, described in section 3.4.1 in more detail, set out to establish the impact of school-based preventive regimes along with the costs of such procedures. At the completion of the study the data were made available to the public, providing access to the most detailed field study on the impact of caries preventive agents yet carried out.

3.4.1: The National Preventive Dentistry Demonstration Programme.

The NPDDP was an extensive project aimed at determining the costs and benefits of various types and combinations of school-based preventive dental care procedures. Funded by the Robert Wood Johnson Foundation, the project ran from 1976 to 1983, with the data being collected over the four- year period 1977-81. The project was conducted in 10 different communities in the United States, 5 non-fluoridated and 5 fluoridated. In each of the communities, 6 different groups

were established: one a control, the remaining 5 receiving differing combinations of preventive regimes. These regimes included fissure sealants, topical fluoride rinses, fluoride tablets, and school based oral health education programmes. The subjects involved in the trial were children in grades 1, 2 and 5 in the participating schools. From those selected as eligible to participate in the study, an 82% response rate was recorded. The data used in this project covers only those children: no attempt has been made to assess the impact of non-responders as the aim of this project is fundamentally different from that of the original study. The background, organisation and results of the programme have been reported extensively in the literature, (Bell et al., 1982; Robert Wood Johnson Foundation, 1983; Klein et al., 1985; Disney et al., 1990).

The project has however been criticised on a number of accounts. In particular, the American Association of Public Health Dentistry (AAPHD) wrote a detailed critical report of the study, (American Association of Public Health Dentistry, 1985). In their response five areas were commented on:

i. issues surrounding the validity of the results. The results of the programme were released to the press prior to any peer review. It was perceived that this could lead to a bias;

ii. monographs on the results were released by the Rand Corporation but their subsequent distribution appears uncertain. This again was perceived as possibly leading to bias reporting;

iii. the monographs themselves did not provide sufficient detail about certain aspects of the methodology used. One example given was the lack of a basis for the allocation of schools to a particular regime;

iv. the cost-effectiveness of the various procedures was not assessed, although this was believed to be a stated aim of the project; and

v. the validity of the programme results were questioned due to faulty research design.

The NPDDP study team subsequently responded to these criticisms, in particular dealing with those concerns which could affect the interpretation of the results regarding cost and effectiveness issues, (Klein et al., 1986). The authors concluded that '..(the) concerns about the NPDDP are based on highly improbable scenarios and purely theoretical considerations. Empirical tests of the potential problems demonstrate they had little or no impact on NPDDP results'.

Due to the differing aims of this and the NPDDP projects the analyses performed vary and the majority of issues raised are of no concern. However, two aspects concerning the examination procedures are worthy of comment. The AAPHD commented that first, differences in decay levels between fluoridated and non fluoridated sites could have been in part due to the examiners' knowledge of the fluoridation status of a site, and secondly, having a child seen by one examiner at baseline and another examiner at the end of the study could have theoretically biased results. Both these issues could affect the subsequent results of this project as it is the presence or absence of decay that is of prime importance. Klein et al. (1986) responded to the two aspects using empirical data. With respect to the impact of knowing whether a site was fluoridated or not, the example of Wichita and Hayward was used. At the commencement of the programme it was believed that Wichita was a non fluoridated area and Hayward fluoridated. Examination of the water supply after the baseline caries data were collected revealed both beliefs to be wrong. Subsequent analyses of the examiners recorded data show no impact of their beliefs on fluoridation status on the recording of caries status.

The second issue, that the examiner:child pairing could influence the caries scores, was also refuted through a two-stage process. The first stage investigated

the extent to which there were systematic differences between examiners within a given year in their tendency to classify a surface as carious, the second stage, the number of examination errors that occurred when pairings were and were not maintained. As with the previous issue, the analyses provided no evidence that the pairing influenced the outcome, and Klein reported:

> '...(these findings) provide unequivocal evidence that the examiner/child pairings had no impact on estimates of regimen effects or the power and precision of the statistical analyses that were conducted.'

3.4.2: The BASCD data sets.

The British Association for the Study of Community Dentistry (BASCD) have been responsible for the organisation, collection, and reporting of levels of caries in 5, 12 and 14 year-old children at a local level since 1985-6. The point prevalence surveys use a clinical examination and employ trained and calibrated examiners, (Palmer et al., 1984).

Three data sets are used from the BASCD surveys: two at a population level, one at an individual level. The two population data sets and those of the 1990-91 14 year-olds survey and the 1992-93 12 year-old survey. The data were collected according to the guidelines produced following a BASCD workshop in 1982, (Palmer et al., 1984), subsequently modified by Dowell and Evans (1988), for the collection of data on the 14 year-olds, and Mitropolous et al. (1992) for the survey of 12 year-olds. The diagnostic threshold for caries used recognised lesions that extend into dentine on the basis of a visual examination only.

The survey of the 14 year-olds report data from nearly 115,000 children grouped into 179 samples, with an average sampling fraction of 0.22. The survey of 12 year-olds covered just over 150,000 children, grouped into 186 samples, with an average sampling fraction of 0.27, (Pitts and Palmer, 1997).

The process of collating the data for BASCD requires each of the local area samples to return only limited information including sample size, and the mean and standard deviation of the components of the DMF index. The data on the individuals in the samples covering Blackpool and Fylde for the 12 year-olds and 14 year-olds surveys were obtained along with three other data sets on 10, 12 and 14 year olds in the same area from surveys carried out in the years 1986, 1988 and 1990. All these data were collected using the standardised BASCD methodology and allow the creation of a longitudinal cohort data set. This is used in the testing of the conceptual model.

3.4.3: The University of Wales and Walsall Health Authority data set.

A longitudinal study of primary school children in Wales commenced in 1991. A sample of 5 year-old children entering school have been examined annually on four occasions. Data on caries experience by tooth surface were collected, again according to the BASCD criteria. As with the BASCD data sets discussed in Section 3.4.2, the data obtained were used in the testing of the conceptual model.

3.5: Data handling

Data analyses were performed on an Alpha mainframe system running UNIX and version 4.1 of SPSS. The data files of the NPDDP were supplied by the Rand

Corporation in ASCII format and subsequently read onto the mainframe system. Two of the five data files were utilised in this project: the master file containing the demographic information of each individual and the clinical file, containing the status of each tooth site.

The data coding used in the NPDDP involved a series of sub-routines to define the presence of a tooth followed by a series of binary codes to define each tooth site. For example, for each tooth one variable was used to describe whether it was present or absent, and for each of the tooth sites three variable were used to describe whether the tooth was decayed, sound, or filled. Thus for each tooth with five surfaces a total of 16 variables were used. For the purpose of this study each tooth surface was coded as either sound or having undergone a caries attack leading to cavitation. Due to the data structure a series of loop and vector routines were programmed into SPSS to make the conversion. These steps resulted in each child's dentition having 128 variables for each year of the study along with the demographic information.

Subsets of the data set were produced and subsequently analysed on a Apple Macintosh running Mac OS 7.5.3 using SPSS v4.0, Deltagraph v3.0, and iThink v2.0.

4. RESULTS

4.1: Introduction

The conceptual model proposed in Section 3 requires a number of hypotheses to be tested. These were:

a. that there was a relationship between the overall mean caries experience and the prevalence of the disease;

b. that there was a relationship between the mean caries score and the associated variance;

c. that there is a relationship between the mean score and the percentage distribution at each DMF score; and

d. that a hierarchy of caries attack exists. At each DMF score the teeth and sites that have been attacked are known.

The results of the analyses of the NPDDP data set are used to define the conceptual model and are presented in five sections. The first section covers the basic demographic details of the study population; the second, the distribution of caries experience; the third the relationship between the mean population caries experience and the variance. The fourth section examines both the inter-population and intra-oral distribution of caries at differing DMF scores, and the final section the impact of various preventive programmes.

These five sections provide the basis for the development of the proposed model concerning caries development and distribution. Most of the data used in the analyses are those collected in the National Preventive Dentistry Programme (NPDDP).

4.2: Demographic characteristics of the NPDDP study population.

The National Preventive Dentistry Programme (NPDDP) was a four year longitudinal programme carried out between 1977 and 1981. It was designed to assess the impact of differing preventative regimes under varying circumstances. The study population used in the study consisted of 24,430 children in 10 differing cities within the United States. There were approximately equal numbers of male and female children, 12,459 and 11,971 respectively. Two thousand and seventeen children joined the programme after the study commenced and are not

Table 1: Demographic characteristics of NPDDP data (white and black)

Location	male black	female black	male white	female white
Chattanooga	104	114	1020	984
Billerica	7	11	1298	1190
New York	137	150	402	385
Minneapolis	52	59	1435	1498
Wichita	162	210	925	839
Pierce County	7	15	1234	1142
Hayward	83	97	718	656
El Paso	42	33	545	471
Monroe	672	658	386	346
Tallahassee	378	333	1036	974

included in the analyses presented here as there are a number of shortcomings in recorded data. Other variables used to describe the population include ethnic

group; white (17484), black (3324), Hispanic (2579) or other (1034), Tables 1 and 2, and information concerning the 10 locations.

The locations can be subdivided into two; fluoridated areas in with fluoride levels of the water supply in the range 0.8 to 1.0 parts per million, or non-fluoridated areas with less than 0.2 parts per million (Table 3).

Table 2: Demographic characteristics of NPDDP data (Hispanic and other)

Location	male Hispanic	female Hispanic	male other	female other
Chattanooga	-	-	9	12
Billerica	1	2	1	3
New York	322	320	361	338
Minneapolis	4	3	32	35
Wichita	18	20	17	13
Pierce County	3	6	12	16
Hayward	251	236	75	66
El Paso	716	669	8	9
Monroe	-	1	3	-
Tallahassee	4	3	12	12

At the time of the first examination the mean age of the programme population was 8.78 years. The age range was between 5.01 and 15.98 years. These ages cover eight school year grades. Over 75% of the children were in 1st, 2nd or 5th

grade at the start of the study. The ages of the study population at the commencement of the programme have been banded and are shown in Table 4.

Table 3: Water fluoride levels for programme sites

Location	Fluoride Status
Chattanooga	0.80 to 1.00ppm
Billerica	less than 0.20ppm
New York	0.80 to 1.00ppm
Minneapolis	0.80 to 1.00ppm
Wichita	less than 0.20ppm
Pierce County	less than 0.20ppm
Hayward	0.80 to 1.00ppm
El Paso	0.80 to 1.00ppm
Monroe	less than 0.20ppm
Tallahassee	less than 0.20ppm

Table 4: Age of participants at commencement of NDPPD study

Age band (years)	number
6.50 or below	389
6.51 to 9.50	14304
9.51 to 12.50	8345
12.51 to 15.50	1750
15.51 or older	12

4.3: Overall distribution of dental caries prevalence and severity

At the commencement of the programme the overall mean DMF-S for the programme participants was 2.43, with a standard deviation of 3.88. At a mean DMF-S of 2.43 the overall caries prevalence was 52.6%. The mean scores for each programme site are shown in Table 5 along with the caries prevalence. By the second year the mean DMF-S score had increased to 2.53, and in subsequent years further increments of 0.68, 0.76 and 0.54 occurred to give a final mean DMF-S score of 4.51 overall. The changes in caries experience by site are shown in Figure 5.

The overall mean DMF-T for the group at the commencement of the programme was 1.59, with El Paso having the lowest mean DMF-T score, 1.01, and Monroe the highest, 2.29, (Table 5).

Table 6 shows the mean DMF-S, DMF-T and prevalence data at the end of the NDPPD study programme with the exception of group based in New York where participation stopped after 3 years. As at the commencement of the programme, Monroe had both the highest mean DMF-T and -S scores, 3.98 and 7.95 respectively, and El Paso the lowest, with a mean DMF-T score of 1.62 and a mean DMF-S score of 2.43.

Figures 6 and 7 illustrates the relationship between the mean DMF-T and DMF-S score and the prevalence through the programme. As the mean DMF score decreases the percentage of people who are defined as caries free, that is have a DMF-S score of 0, increases. The reverse is also true. As the prevalence increases, the mean DMF-S also increases. This changing relationship between the mean DMF-S score and prevalence is not limited to a sub-group of the population. There would appear to be a population movement. As the mean DMF

Figure 5: The change in caries experience by NPDDP programme site

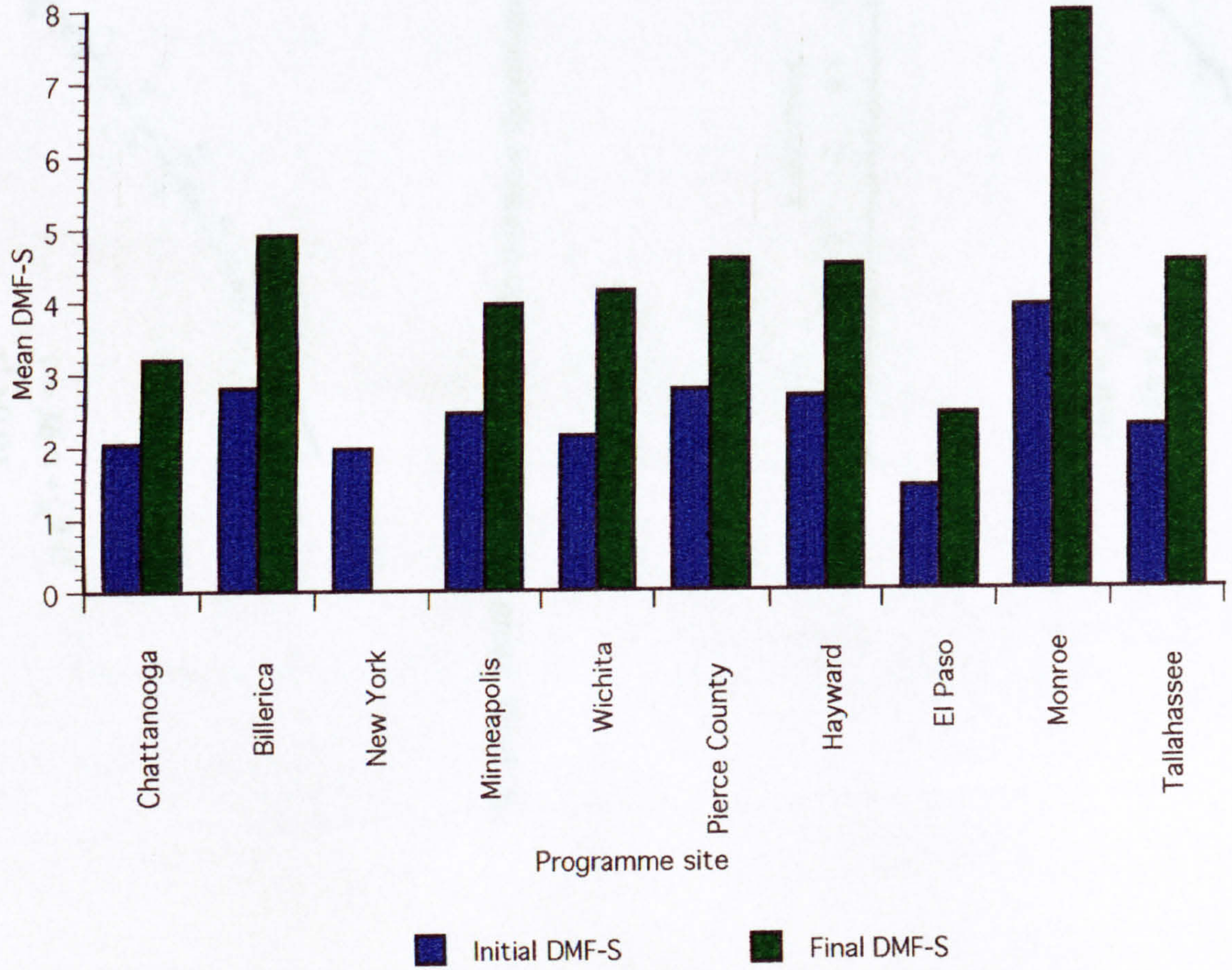


Figure 6: The relationship between caries prevalence and mean DMF-T.

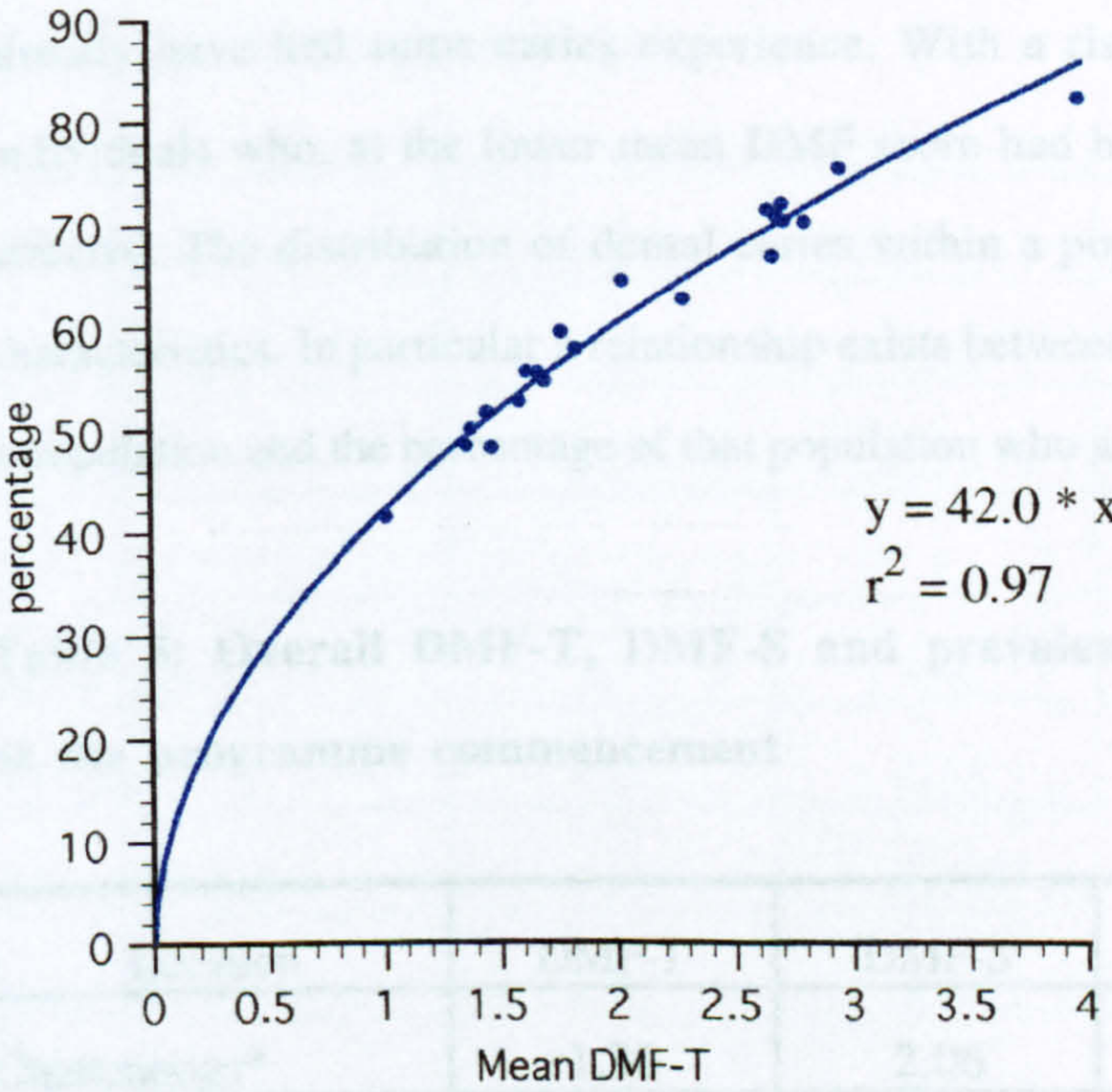
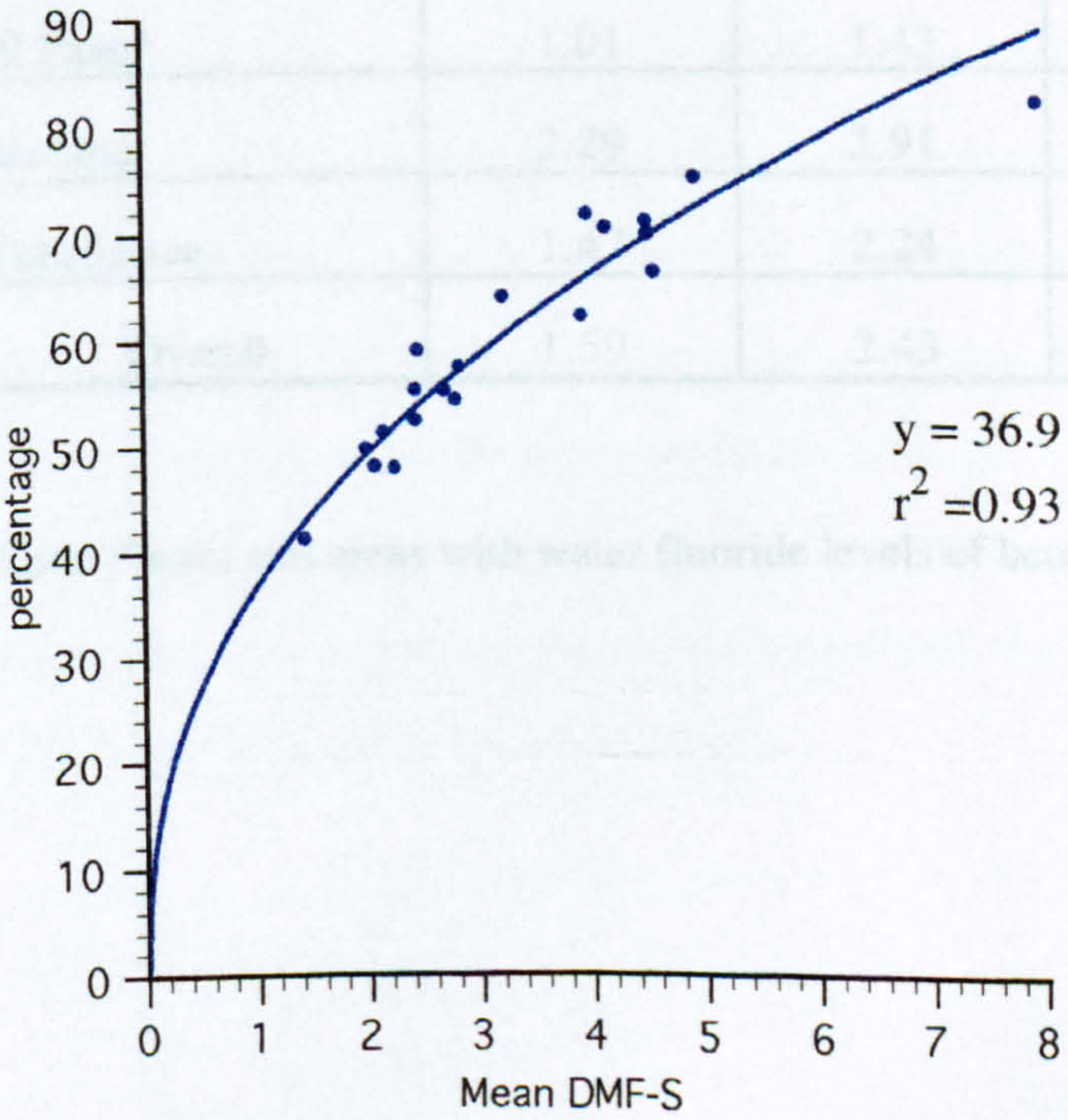


Figure 7: The relationship between caries prevalence and mean DMF-S.



score rises, the increase is not confined to a sub-group of the population who already have had some caries experience. With a rise in caries levels those individuals who, at the lower mean DMF score had been caries free, are also affected. The distribution of dental caries within a population exhibits certain characteristics. In particular a relationship exists between the mean DMF score of a population and the percentage of that population who are caries free.

Table 5: Overall DMF-T, DMF-S and prevalence data for each site at the programme commencement

Location	DMF-T	DMF-S	prevalence
Chattanooga*	1.36	2.06	48.3
Billerica	1.83	2.82	57.7
New York*	1.38	1.98	49.9
Minneapolis*	1.77	2.45	59.3
Wichita	1.45	2.14	51.5
Pierce County	1.70	2.78	54.5
Hayward*	1.67	2.68	55.4
El Paso*	1.01	1.43	41.4
Monroe	2.29	3.91	62.5
Tallahassee	1.47	2.24	48.1
Overall	1.59	2.43	52.6

Note: * indicates areas with water fluoride levels of between 0.8 and 1.00ppm .

Table 6: Overall DMF-T, DMF-S and prevalence data for each site at the end of the programme

Location	DMF-T	DMF-S	prevalence
Chattanooga	2.03	3.21	35.7
Billerica	2.96	4.92	24.6
New York	-	-	-
Minneapolis	2.71	3.96	28.1
Wichita	2.69	4.13	29.3
Pierce County	2.67	4.56	33.3
Hayward	2.65	4.49	28.7
El Paso	1.62	2.43	44.5
Monroe	3.98	7.95	17.7
Tallahassee	2.81	4.50	29.9
Overall	2.72	4.51	29.8

Any distribution has limits; at one end will be those individuals with a low score, at the opposite, those with a high score. When reporting caries distribution within a population, those individuals with lower than average caries experience will be found at the left hand of the distribution, those with higher than average at the right hand side. The percentage caries free are found at the left hand side of any distribution, and, if the absolute magnitude of the left hand tail of the distribution is correlated with the mean, the question must be asked what are the effects of changing caries experience on the right hand tail, that is those individuals who have a higher than average level of caries.

Figures 8 and 9 show the relationship between mean DMF-S score and the percentage of a distribution with a DMF-S score of 10 or more and 30 or more respectively. In both cases as the mean DMF-S score decreases, the tail which contains a so called high-risk group also decreases. For example, at a mean score of 7 one would expect to find 23.5% of the population with a DMF-S score equal to or greater than 10, and 1.13% with a DMF-S score equal to or greater than 30. If the mean DMF-S score is equal to 2, only for 1.17% of the population would be expected to have a DMF-S greater than or equal to 10, and no individual a DMF-S score greater or equal to 30.

The next question to ask is whether the population shift is a constant, i.e. as the mean increases, do all members of the population experience a similar effect, or are the effects unequally distributed? This question can be answered by looking at the distribution around the mean. If the variance remains the same, then the effects of any change will be distributed equally among the population. If the variance increases with the mean, then the gap between those at one end of the tail and the other will widen. Figure 10 shows the relationship between the variance and the mean DMF-S for the NDPPD data set. As the mean increases the variance also increases. The distribution of caries experience widens within the population as the mean increases, but in addition the impact is not confined to a sub-group: the increase is not confined to those with caries initially.

These two important findings in alterations in caries distribution according to prevalence will be fundamental for defining a strategy. First, as the mean increases, the effect is felt throughout the population. This would challenge the frequently stated concept that there is either a sub group of the population which is at either high risk or low risk of the development of dental caries. When the prevalence of caries increases everybody increases their propensity of developing new carious lesions; the risk of any member of the population developing a

Figure 8: The relationship between mean DMF-S score and the percentage of a distribution with a DMF-S score of 10 or more

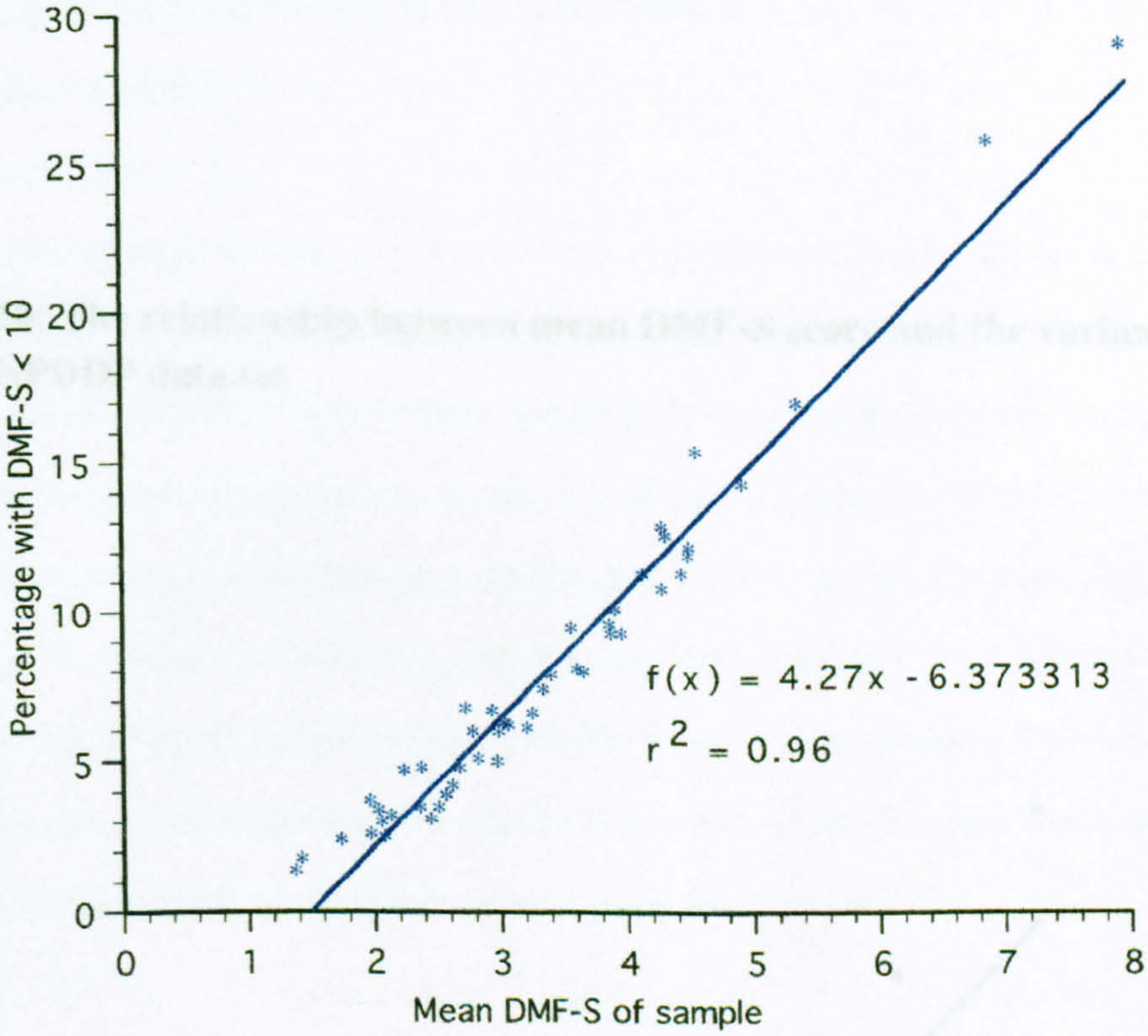


Figure 9: The relationship between mean DMF-S score and the percentage of a distribution with a DMF-S score of 30 or more

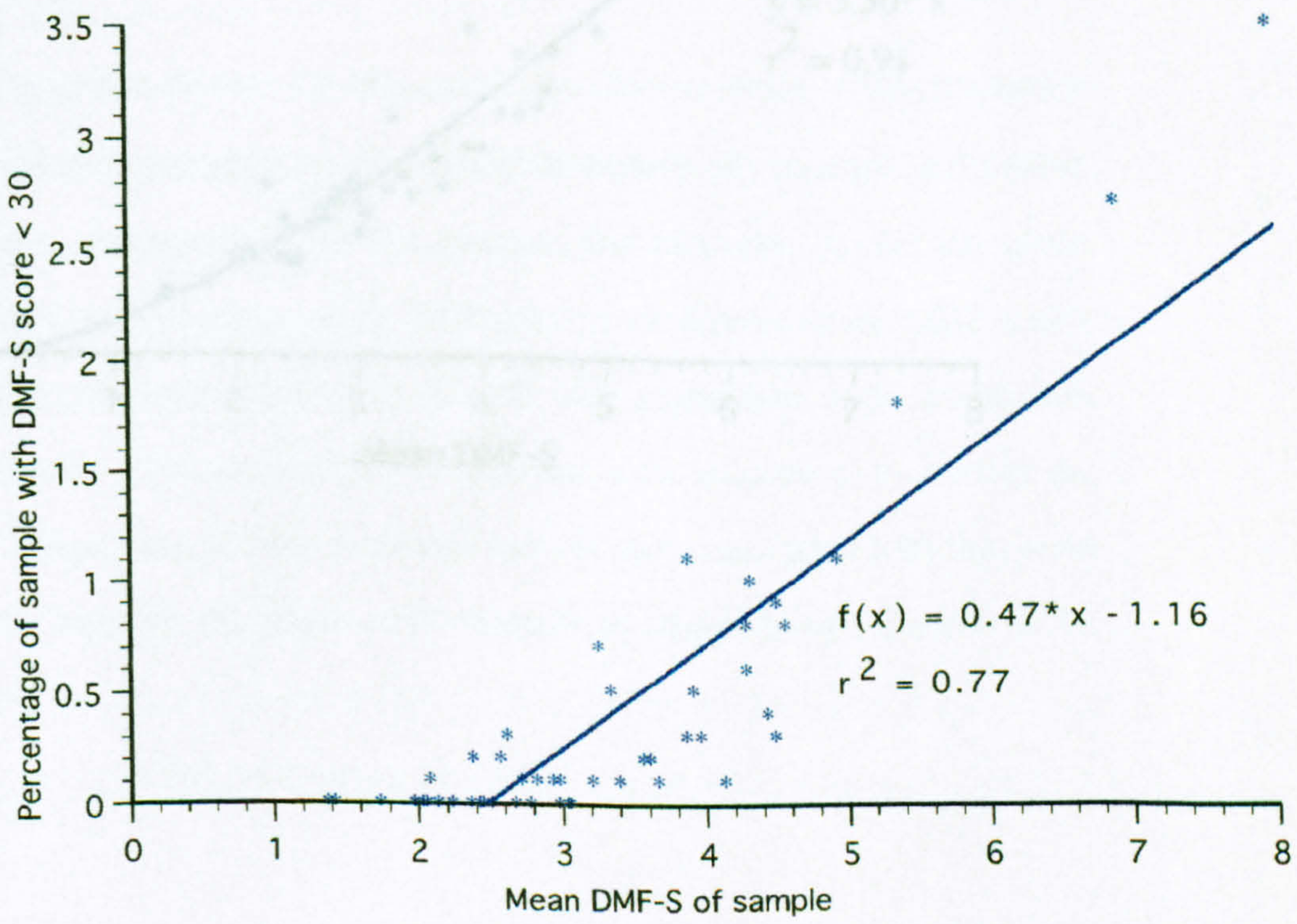
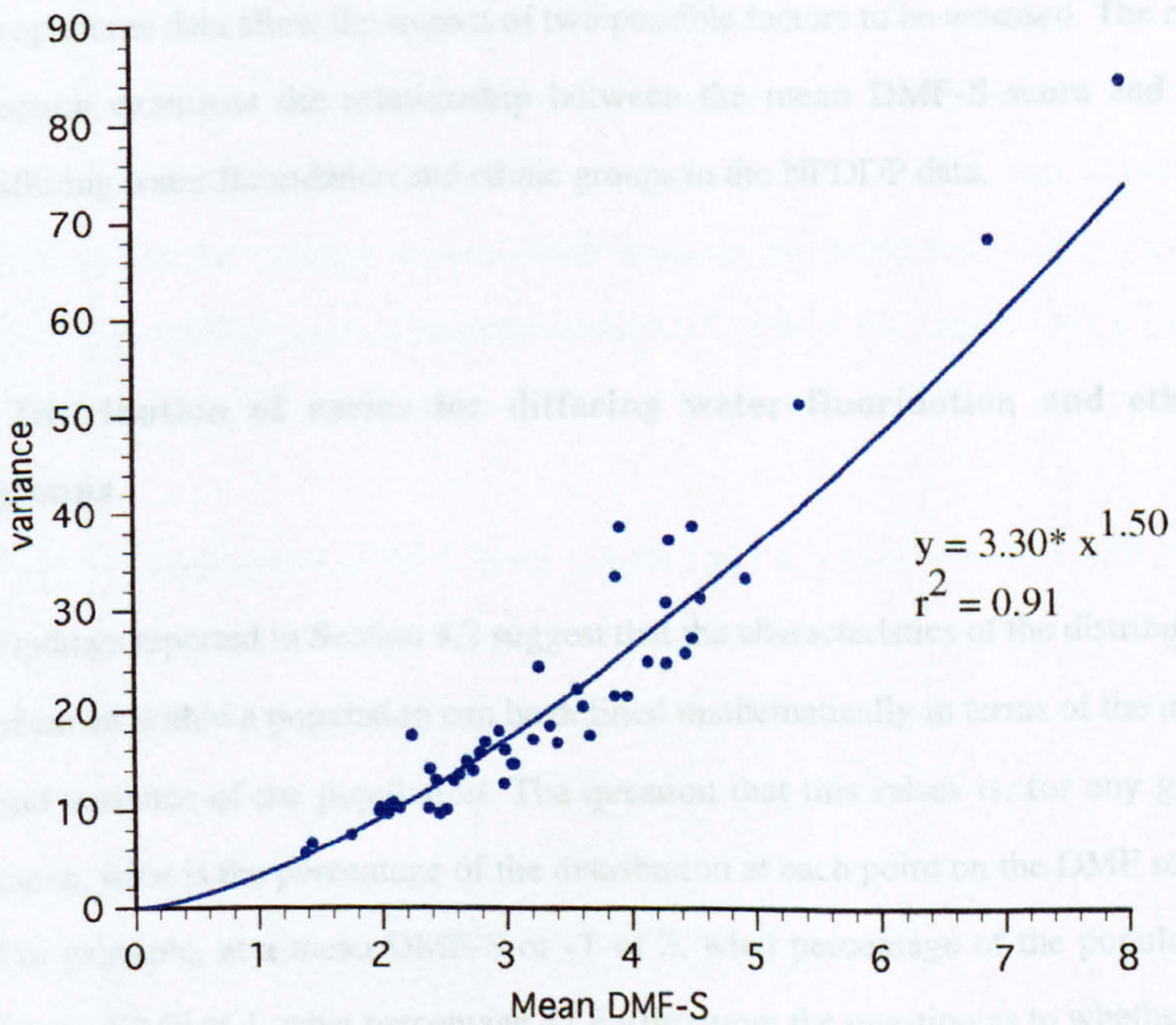


Figure 10: The relationship between mean DMF-S score and the variance for the NPDDP data set



carious lesion increases. Although there is still a group of individuals who are defined as being caries free - of having a DMF score of 0 - the DMF index is insufficiently sensitive to be able to detect any change in risk as they have not experienced cavitation.

Secondly, this change in risk is not equal for a given population. There is a widening in the variance, that is the distribution of the disease. For a population there is an overall increase, but for some members of the population this increase is greater than others. This poses the question of the nature of the development in risk; are there factors within the population that would explain the changing relationship between the variance and mean DMF-S score? The NPDDP programme data allow the impact of two possible factors to be assessed. The next section examines the relationship between the mean DMF-S score and the differing water fluoridation and ethnic groups in the NPDDP data.

4.3.1: Distribution of caries for differing water fluoridation and ethnic groups

Findings reported in Section 4.2 suggest that the characteristics of the distribution of caries within a population can be defined mathematically in terms of the mean and variance of the population. The question that this raises is, for any given mean, what is the percentage of the distribution at each point on the DMF scale? For example, at a mean DMF-S or -T of 2, what percentage of the population have a DMF of 1, what percentage 4? Furthermore the question as to whether the pattern is independent of other variables, such as those associated with the caries process, for example the presence of fluoride or ethnic grouping needs to be answered.

Figures 11 and 12 show the relationship between mean DMF-S scores and caries prevalence during the NPDDP for water fluoridation and for the differing ethnic groupings. As with geographical site, the determining factor in the distribution appears to be the overall DMF-S score. For a given DMF-S, the prevalence in non-fluoridated and fluoridated areas is similar.

In the NDPPD study, Monroe, a non-fluoridated area, had a DMF-S of 3.91 at the commencement of the study. Tallahassee, also a non-fluoridated area, had a DMF-S score of 3.88 recorded in the fourth year of the study, and Minneapolis, a fluoridated area, a DMF-S of 3.96 recorded in the final year of the programme. Monroe has the highest proportion of black children in the study population, nearly 60%. This is far greater than both Tallahassee or Minneapolis, (Table 1). Figure 11 shows the distribution of the percentage of each population according to their DMF-S score. Although there is some variation, which may in part be explained by the variation in mean DMF-S scores, the distribution is very similar. At a mean DMF-S score of approximately 4.00, 10% of the sample have a DMF-S of 1, and a similar percentage a DMF-S of 2.

The percentage with a DMF-S score of both 3 and 4 is approximately 7.5%, with no major variation between the areas. As the DMF-S increase the percentage at each DMF-S value decreases, but importantly, the pattern is very similar for all the areas. Thus it appears that the distribution of the DMF-S of individuals within a population is not dependant upon the level of water fluoridation, ethnic group, or, as the areas are from differing stages of the NPDDP study, the age of individuals.

The above pattern exists for a mean DMF-S of nearly 4.0. Does it exist for other levels of caries? Figure 12 shows the distribution for Chattanooga in year 1 of the study, New York in year 2, and El Paso in year 4. The study population for

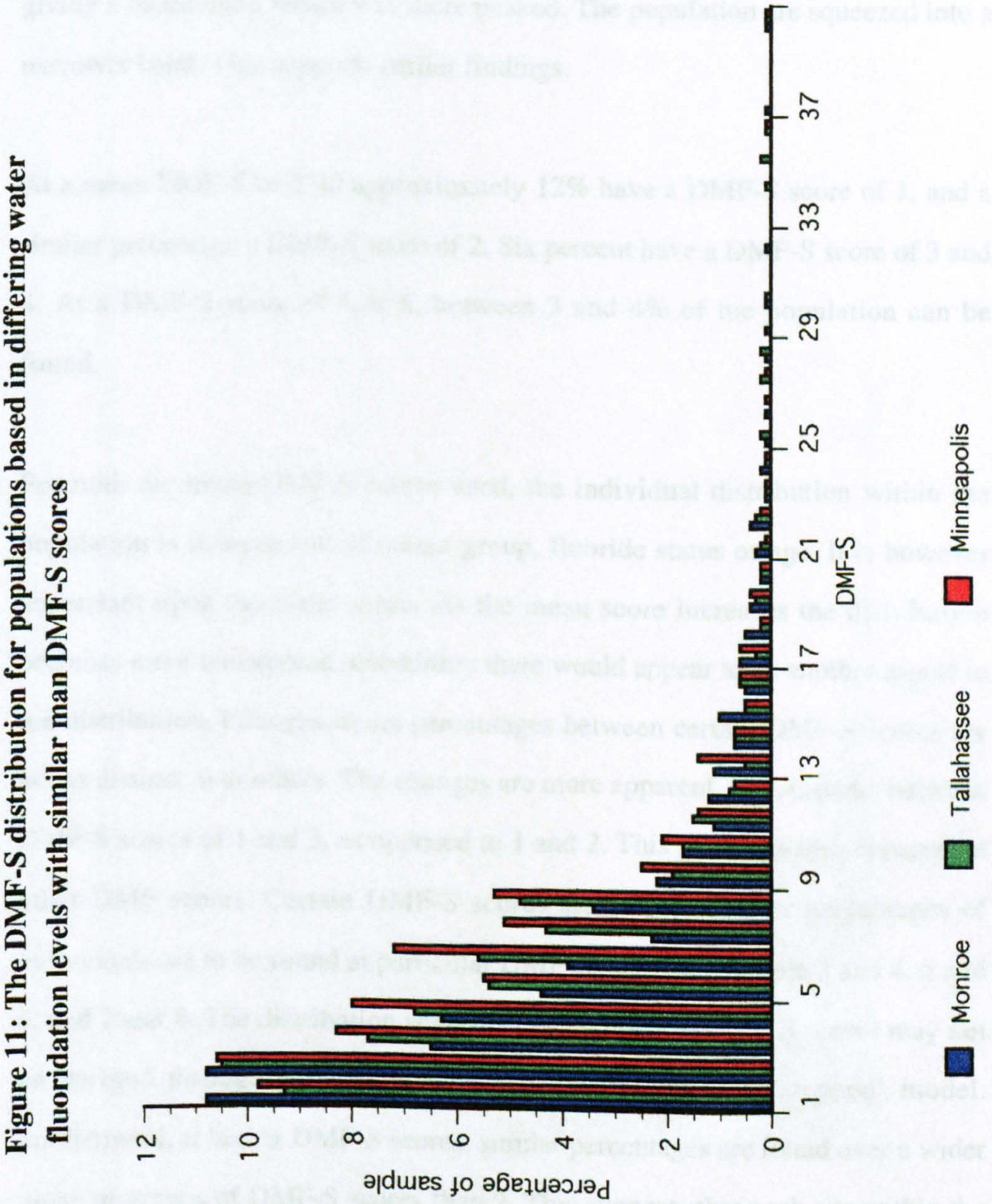


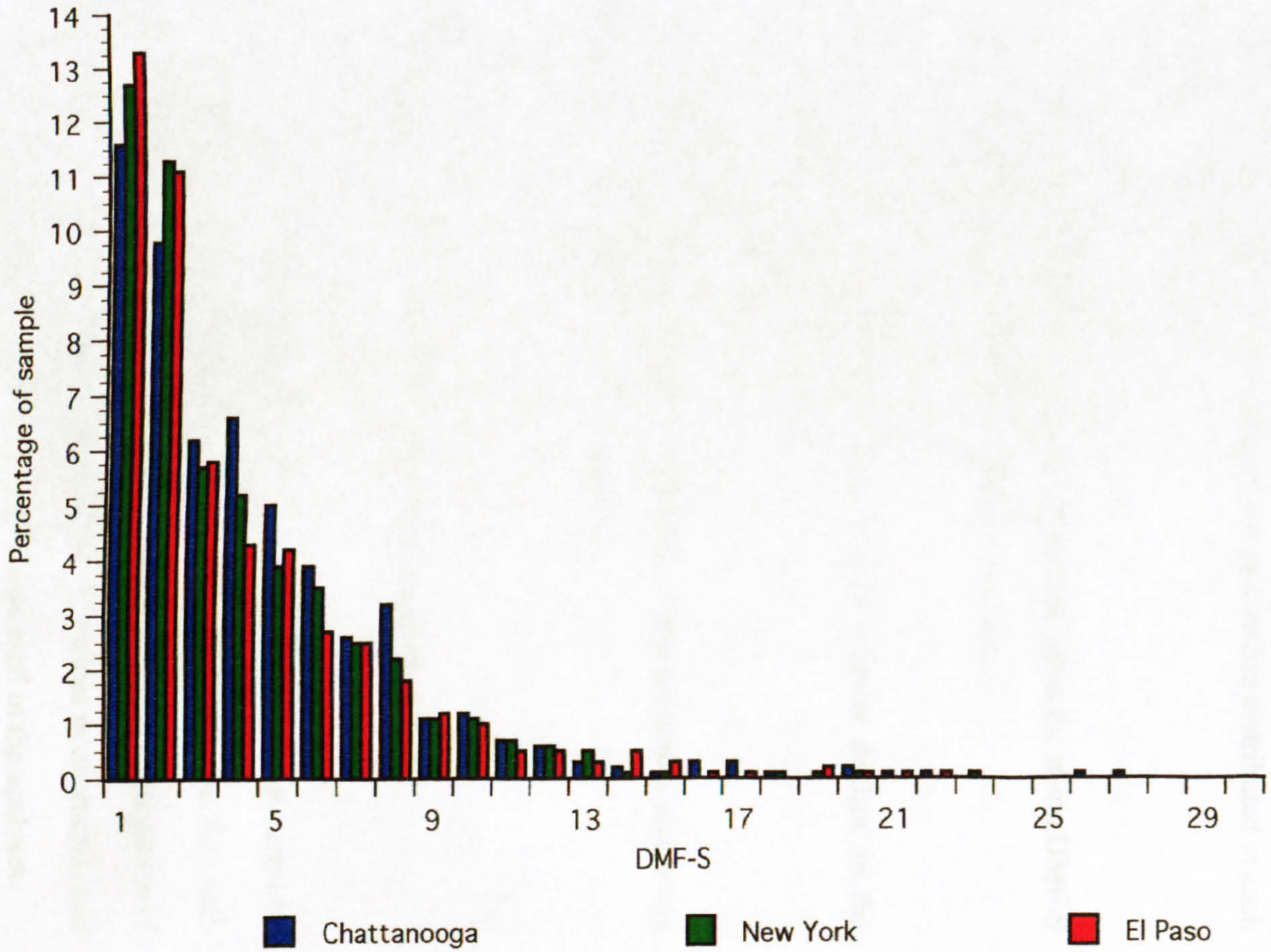
Figure 11: The DMF-S distribution for populations based in differing water fluoridation levels with similar man DMF-S scores
Percentage of sample
0
2
4
6
8
10
12
1
5
9
13
17
21
25
29
33
37
DMF-S
Monroe
Tallahassee
Minneapolis

these areas had a recorded mean DMF-S of about 2.0. Although all areas had a water fluoride level of between 0.8 and 1.0ppm, there were significant differences in the ethnic structure of the groups, (Tables 1 and 2). As with the higher DMF-S all 3 areas exhibited a similar distribution of DMF-S scores within the population. With the reduction in the mean, the variance also decreased, giving a distribution which was more peaked. The population are squeezed into a narrower band. This supports earlier findings.

At a mean DMF-S of 2.00 approximately 12% have a DMF-S score of 1, and a similar percentage a DMF-S score of 2. Six percent have a DMF-S score of 3 and 4. At a DMF-S score of 5 or 6, between 3 and 4% of the population can be found.

For both the mean DMF-S scores used, the individual distribution within the population is independent of ethnic group, fluoride status or age. It is however dependant upon the mean score. As the mean score increases the distribution becomes more widespread. In addition there would appear to be another aspect to the distribution. Changes in the percentages between certain DMF-S scores are not as distinct as at others. The changes are more apparent, for example, between DMF-S scores of 1 and 3, as opposed to 1 and 2. This pattern is also apparent at other DMF scores. Certain DMF-S scores are paired. Similar percentages of individuals are to be found at particular DMF scores, for example 3 and 4, 5 and 6, and 7 and 8. The distribution suggests that changes in DMF-S scores may not be defined through a linear progressive model, but as a 'stepped' model. Furthermore, at higher DMF-S scores, similar percentages are found over a wider range of groups of DMF-S scores than 2. This suggests that each site within the mouth may not be totally independent. Sites may have different probabilities of susceptibility to caries attack.

Figure 12: The DMF-S distribution for differing ethnic population structures with similar man DMF-S scores



The results in this section would suggest that caries distribution within the population can be modelled. The data indicate that there is a relationship between the prevalence and the severity of dental caries. As the prevalence increases, the severity of caries within the population also increases. The key features of the model are:

i. for a given mean DMF-S the percentage of the population distributed at each DMF-S interval could be defined.

ii. The percentage at each DMF-S interval is dependant upon the mean DMF-S score but independent of fluoride status, ethnicity and age.

iii. There appears to be a stepped as opposed to a linear decline in the percentage found at each DMF-S interval.

To further test this latter hypothesis, the probability of any particular site being carious for a given DMF-S needs to be ascertained.

4.4: The probability of individual tooth sites undergoing attack

In this section the results of the analyses concerning the distribution of caries by tooth site are reported. The probabilities derived are for each site for each individual and are then aggregated for the sample population. The aggregation of probabilities gives rise to a distribution, approximately normal in character, and the mean of this distribution is subsequently reported and used in the analyses.

The probability of an event occurring ranges from zero to 1. Obviously, as the overall DMF score rises, the probabilities of any site being carious will change.

For example, if a group of 128 individuals, each having a DMF-S score of 1 and all sites exhibited the same propensity for caries, it would be expected that the distribution of sites affected within the mouth would be random, the probability for a particular site being carious being 1/128. If, however, the group all had a DMF-S of 128, the relative probability of finding a particular site carious would remain the same, but this time the absolute probability would have changed from 1/128 to 1.

As the DMF score increases, the probabilities alter. However, once a probability of 1 has been reached no further increase is possible. Thus, when examining changes in the distribution of caries at different DMF-S scores, the ratio between individual probability scores is not important, only the overall ranking that is exhibited by the probabilities for each site. The order of susceptibly will be determined by the relative values of the probabilities. Whether an individual site is twice as likely to become carious as another, cannot be determined using this approach. However, it may be that certain sites exhibit similar probabilities; for example a particular site on the left hand side of the mouth may have a similar mean probability as the corresponding site on the right hand side, or that other factors may influence the distribution of probabilities, for example it has been suggested that fluoride has a more beneficial impact on certain sites than others.

The probabilities presented in this section are analysed by a number of variables; sex, ethnicity, level of fluoride and age. For each variable the probability of a particular site being found carious at a given DMF score was calculated. A Spearman ranking test was then carried out in order to assess whether the relative order of site susceptibility was independent of the particular variable. As previously, the data used in this section are from the NPDDP data set.

4.4.1: The probability of particular tooth sites being carious by demographic variables

For each tooth site the probability of the site being carious was calculated, and the sites ordered according to their individual probabilities. The most likely site being ranked first, the least, last. The resulting data sets were then analysed by various demographic factors.

4.4.1.1: Tooth site probability by sex

The correlation between the site probabilities for the study sample according to sex was 0.83 using the Spearman ranking test, with a level of significance less than 0.001. This indicates that sex was not an important determinant of a tooth site's susceptibility to caries.

4.4.1.2: Tooth site probability by ethnicity

Table 7 shows the correlation between the site probabilities for the differing ethnic groups involved in the NPDDP study. The column and rows are the ethnic groupings and the matrix produced represents the correlation co-efficient for each of the sub groups. For example, the correlation between the black and white groups was 0.8527, between Hispanic and white was 0.8275, and white and other 0.8538.

All groups when compared to each other show a strong correlation ranging from over 0.91 for the Hispanic and black groups, to 0.82 for the Hispanic and white groups. The level of significance for all of the comparisons was greater than 0.001.

Table 7: Spearman correlation matrix for sites by ethnicity

Ethnicity			
black	0.8527		
Hispanic	0.8275	0.9145	
other	0.8538	0.9103	0.9077
	white	black	Hispanic

significance: p> 0.001

4.4.1.3: Tooth site probability of being carious by level of fluoride

Table 8 shows the correlation between the tooth site probabilities for the differing fluoride levels.

Table 8: Spearman correlation matrix for tooth sites by fluoride level.

Fluoride level		
0-8-1.00ppm	0.8095	
not known	0.8313	0.9048
	less than 0.2ppm	0.8-1.00ppm

significance: p> 0.001

As with the previous correlations the level of significance for all the comparisons was greater than 0.001, and a very high correlation was evident between all three fluoride level groups.

4.4.1.4: Tooth site probability of being carious by programme area.

Table 9 shows the correlation between the site probabilities for the differing programme areas.

As with the previous analyses relating to sex, ethnicity and fluoride the results indicate that the order of susceptibility of tooth sites was similar for each of the programme site.

4.4.1.5: Tooth site probability of being carious by age.

At the commencement of the NPDDP programme the age range of the participants was 5.01 to 15.98 years. To derive the Spearman correlation co-efficients the ages of the participants have been banded, and the mid-point used as the group identifier. For example, the group represented by the age5.5 band consists of all those children below the age of 6 years of age at the date of the examination, the group age6.5 consists of all those aged 6 years or older but less than 7 years of age. Table 10 lists the correlation between the site probabilities for the differing age groups.

The correlations between the differing age groups show both the lowest, and hence the greatest variation, when compared to the other analyses presented, although all remain significant at $p > 0.0001$. This can be explained by the differing number of sites that are erupted in differing age groups. The ranking analysis can only assess the correlation between sites present. Sites that are unerupted, and hence have a probability of zero of being carious, will be ranked at random.

Table 9: Spearman correlation matrix for tooth sites by programme site.

Site									
Tallahassee	0.8202								
Chattanooga	0.7637	0.8004							
Billerica	0.8304	0.7718	0.8132						
New York	0.8201	0.8563	0.7899	0.8124					
Minneapolis	0.7911	0.8469	0.8345	0.8187	0.7862				
Wichita	0.7793	0.7922	0.8045	0.7897	0.8231	0.7745			
Pierce County	0.7537	0.8525	0.8061	0.7777	0.8151	0.8292	0.8185		
Hayward	0.7637	0.8004	0.8153	0.8202	0.7514	0.7311	0.8047	0.8152	
El Paso	0.8303	0.8380	0.7490	0.7816	0.8482	0.8107	0.8077	0.8086	0.8256
	Monroe	Tallahassee	Chattanooga	Billerica	New York	Minneapolis	Wichita	Pierce County	Hayward

Significance: p>0.001

Table 10: Spearman correlation matrix for tooth sites by age group

Site										
age6.5	0.5109									
age7.5	0.4852	0.6761								
age8.5	0.4480	0.5175	0.6598							
age9.5	0.4366	0.5656	0.6885	0.7202						
age10.5	0.3929	0.5903	0.6760	0.7023	0.8488					
age11.5	0.3842	0.5760	0.6488	0.7239	0.8510	0.9119				
age12.5	0.3844	0.5215	0.6324	0.6989	0.8289	0.8795	0.8988			
age13.5	0.3738	0.5295	0.6101	0.6477	0.7815	0.9034	0.8867	0.9004		
age14.5	0.3714	0.4768	0.5858	0..6644	0.7634	0.8631	0.8639	0.8347	0.9110	
age15.5	0.5059	0.5728	0.5427	0.4816	0.6650	0.6235	0.6288	0.6263	0.6319	0.6300
	age5.5	age6.5	age7.5	age8.5	age9.5	age10.5	age11.5	age12.5	age13.5	age14.5

Significance: p>0.001

4.4.2: The order of tooth site susceptibility to dental caries.

In the previous section, the results of the ranking analyses suggest that the order in which tooth surfaces became carious was independent of the variables under study. The order in which surfaces become carious would appear to be constant. The next question that needs to be asked is whether there are groups of sites which become carious for a given attack intensity rather than a single site having a resistance factor, that is two or more sites have similar factors.

Figure 13 shows the distribution of probabilities grouped into 5 categories. The categories were formed by grouping sites with similar probabilities together. The most susceptible sites with defined as having a probability of being carious within the range 0.34 to 0.23, the next group 0.18 to 0.04, then 0.03 to 0.01, then 0.008 to 0.002 and, finally, the least susceptible sites formed the remaining group.

The pattern that evolves indicates that a left:right side symmetry exists, the propensity of attack is similar for the differing sides of the mouth. Furthermore there is a degree of symmetry between the upper and lower jaws, although this is only apparent in the posterior sextants. For the anterior sextants the upper jaw is more prone to attack than the lower jaw.

Figure 14 combines the findings shown in Figure 13 with the developmental patterns by tooth surface type with the probability of finding a particular surface type carious at each DMF score being converted to a ratio. At a DMF-S of 1 only 1 lesion will occur, at a DMF-S score of 5, five lesions will. To allow comparison, the probability of each surface type has been calculated, the total for each DMF-S score being equal to one. As the DMF score increases, the ratio of smooth to approximal to pit and fissured surfaces changes, although for the lower

Figure 13: Distribution of probabilities of site susceptabilities

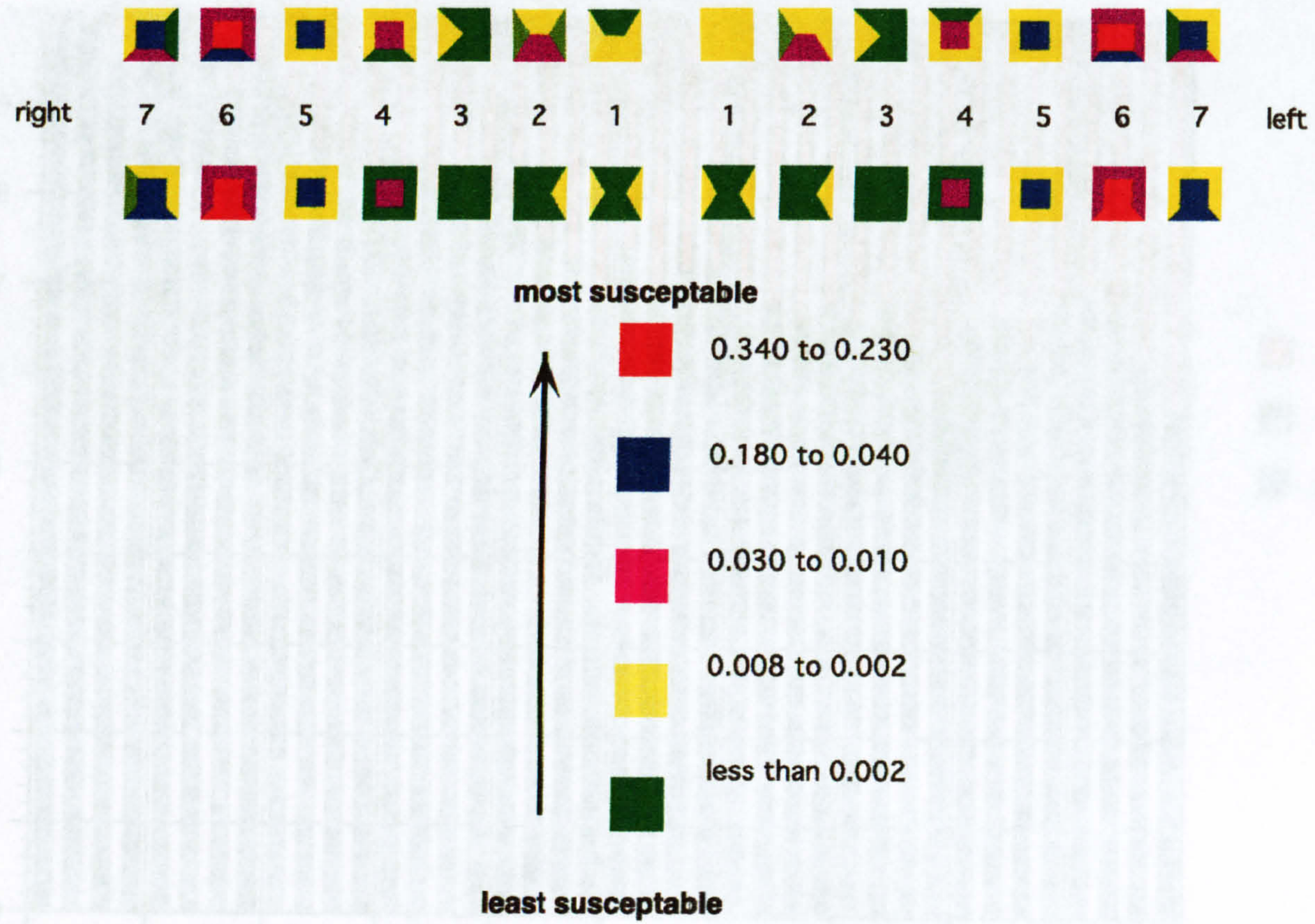


Figure 14: Proportion of each tooth surface type affected by caries at each DMF-S score.

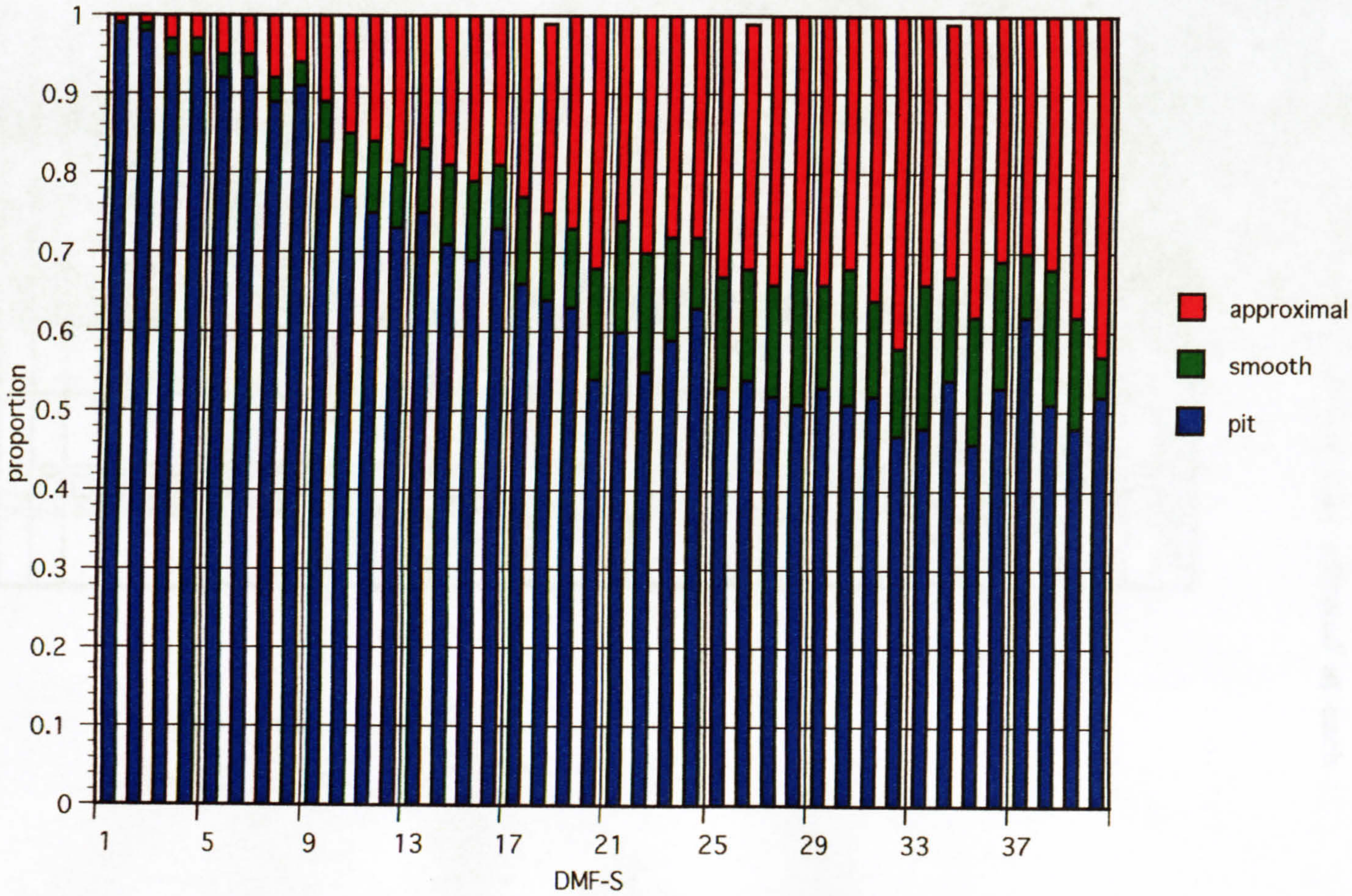


Table 11: Proportion of each tooth surface type affected at each DMF-S score

DMF-S	pit	smooth	approximal
1	0.99	0.00	0.01
2	0.98	0.01	0.02
3	0.95	0.02	0.04
4	0.95	0.02	0.04
5	0.92	0.03	0.05
6	0.92	0.03	0.05
7	0.89	0.03	0.08
8	0.91	0.03	0.07
9	0.84	0.05	0.11
10	0.77	0.08	0.15
11	0.75	0.09	0.16
12	0.73	0.08	0.19
13	0.75	0.08	0.17
14	0.71	0.10	0.19
15	0.69	0.10	0.22
16	0.73	0.08	0.19
17	0.66	0.11	0.24
18	0.64	0.11	0.24
19	0.63	0.10	0.27
20	0.54	0.14	0.33
21	0.60	0.14	0.27
22	0.55	0.15	0.30
23	0.59	0.13	0.28
24	0.63	0.09	0.28
25	0.53	0.14	0.33
26	0.54	0.14	0.31
27	0.52	0.14	0.34
28	0.51	0.17	0.32
29	0.53	0.13	0.34
30	0.51	0.17	0.32
31	0.52	0.12	0.36
32	0.47	0.11	0.42
33	0.48	0.18	0.34
34	0.54	0.13	0.32
35	0.46	0.16	0.38
36	0.53	0.16	0.31
37	0.62	0.08	0.30
38	0.51	0.17	0.32
39	0.48	0.14	0.38
40	0.52	0.05	0.43

DMF scores, any major change in the ratios does not occur until around a DMF-S of 8. Only then does the contribution of approximal or smooth surface type lesions have an impact, (Table 11).

The key features of tooth susceptibility are:

i. there is variation between sites with respect to its susceptibility to caries attack. Certain sites are more prone to caries attack than others.

ii. There is a degree of symmetry. In the vertical plane, the sites show a similar propensity for caries attack: one would expect to find an equal number of lesions on the left hand side of the mouth as on the right. There is an order to the susceptibility: the posterior molar teeth, are more prone than the anterior teeth.

4.4.3: Impact of water fluoridation on tooth site susceptibility to caries.

Figures 15 to 19 show the effects of differing levels of water fluoride on caries on the distribution of caries according to site type at differing age groups. For each sample, the number of sites affected were divided into three surface types; occlusal, buccal and approximal. The total number of each surface type was divided by the number of individuals at each DMF-S and the probabilities of a site type being carious calculated.

At 6 years of age, the level of DMF is low, and in consequence the range of the graph is limited to a maximum of DMF-S of 8, (Figure 15). At a DMF-S of 1 the site types involved are occlusal or buccal only, with similar probabilities. There is a steady increase in occlusal and buccal surface involvement as the DMF-S

increases, with a similar pattern of development irrespective of water fluoride levels.

The above findings are also found at differing ages, (Figures 16 to 19). At ages 8, 10, 12 and 14, the surface type involvement at low DMF-S scores is occlusal or buccal, and major degree of approximal surface type involvement only occurring at a DMF-S score of 8. For all ages the effect of water fluoridation level on the ratios of surface types to DMF-S score is negligible suggesting that the action of fluoride is not surface specific but has an overall effect on caries attack intensity.

4.4.4: Summary of the distributive properties of caries and tooth and site susceptibility to carious attack

The distribution of dental caries within the mouth follows certain patterns. There is a relationship between the mean levels of caries and the percentage caries free. As the mean caries score rises the percentage caries free falls. Furthermore the distribution is not confined to certain sub groups within a study population. As the mean caries score increases, the population experiences an increase in the propensity of developing new carious lesions. The increase in the development of new lesions follows certain patterns: the molar teeth will be the first teeth to undergo carious attack, the lower anterior teeth the least likely. This development in the pattern of carious lesions is independent of age, sex, ethnicity or fluoride.

These results indicate that the development of an accurate caries progression model is possible. The model indicates that the distribution of caries was normal in shape, with a mathematical relationship between the mean and variance. Furthermore the order of both tooth and site susceptibility can be modelled.

Figure 15: Effect of water fluoridation on caries distribution according to tooth site type at 6 years of age

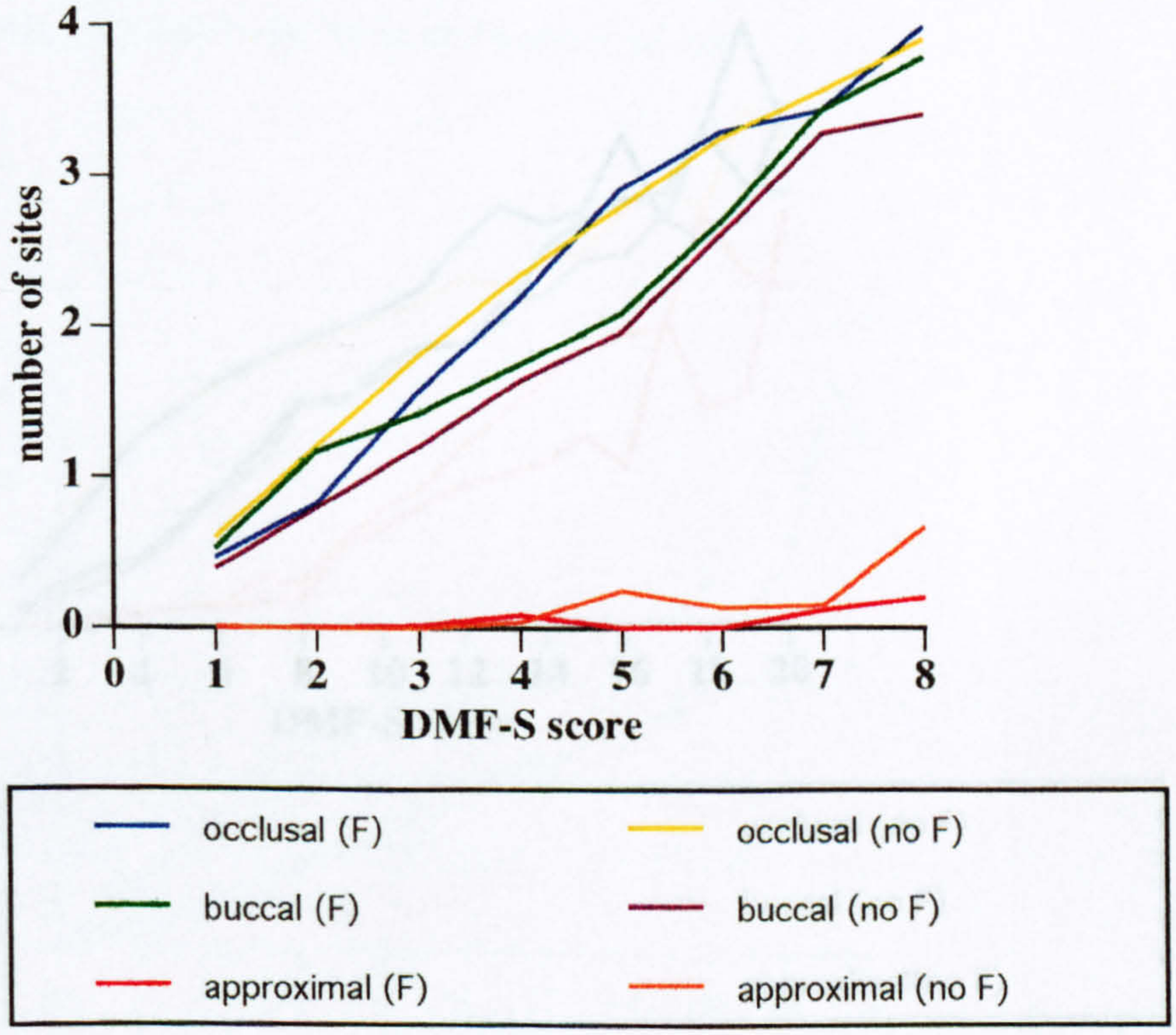


Figure 16: Effect of water fluoridation on caries distribution according to tooth site type at 8 years of age

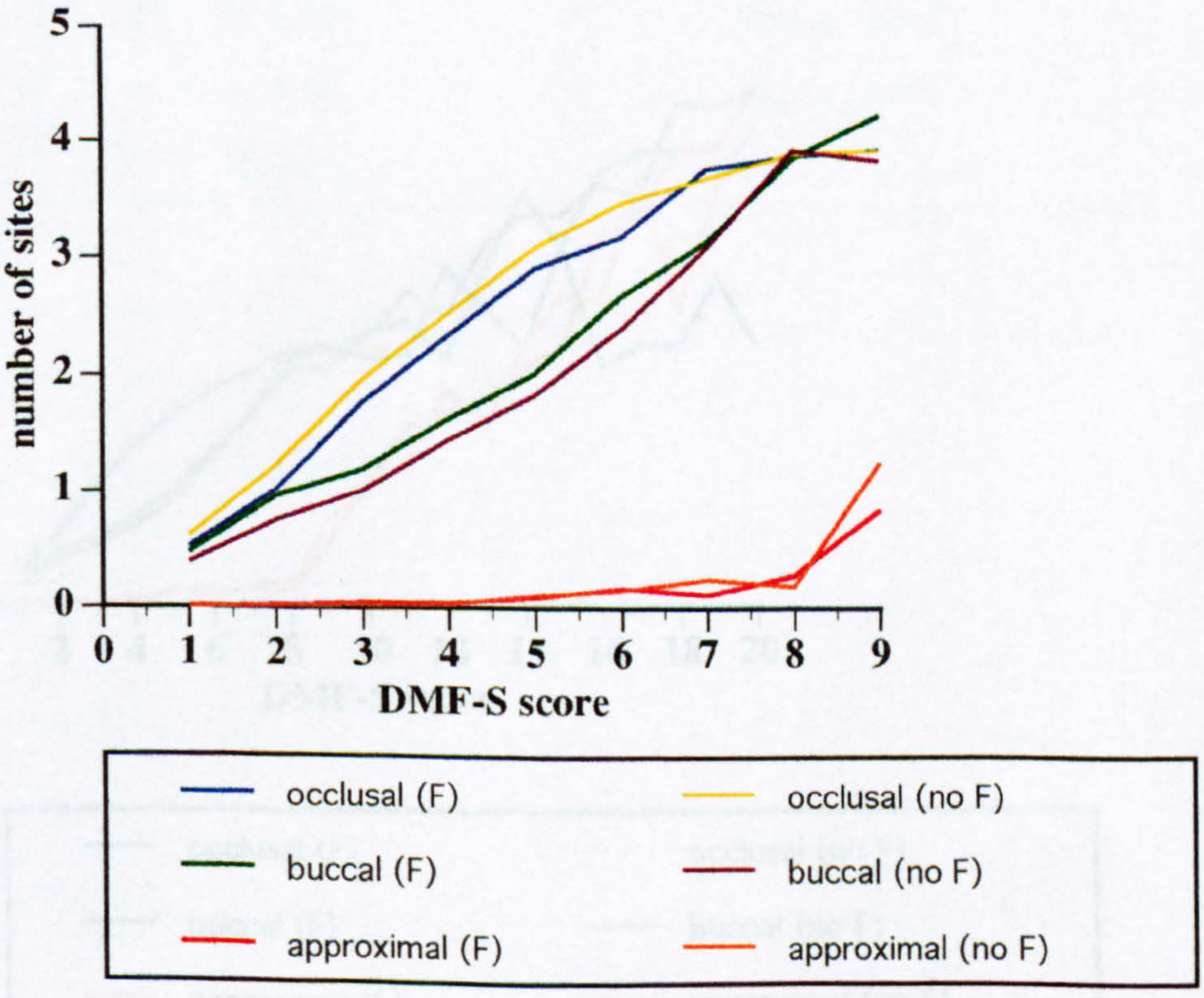


Figure17: Effect of water fluoridation on caries distribution according to tooth site type at 10 years of age

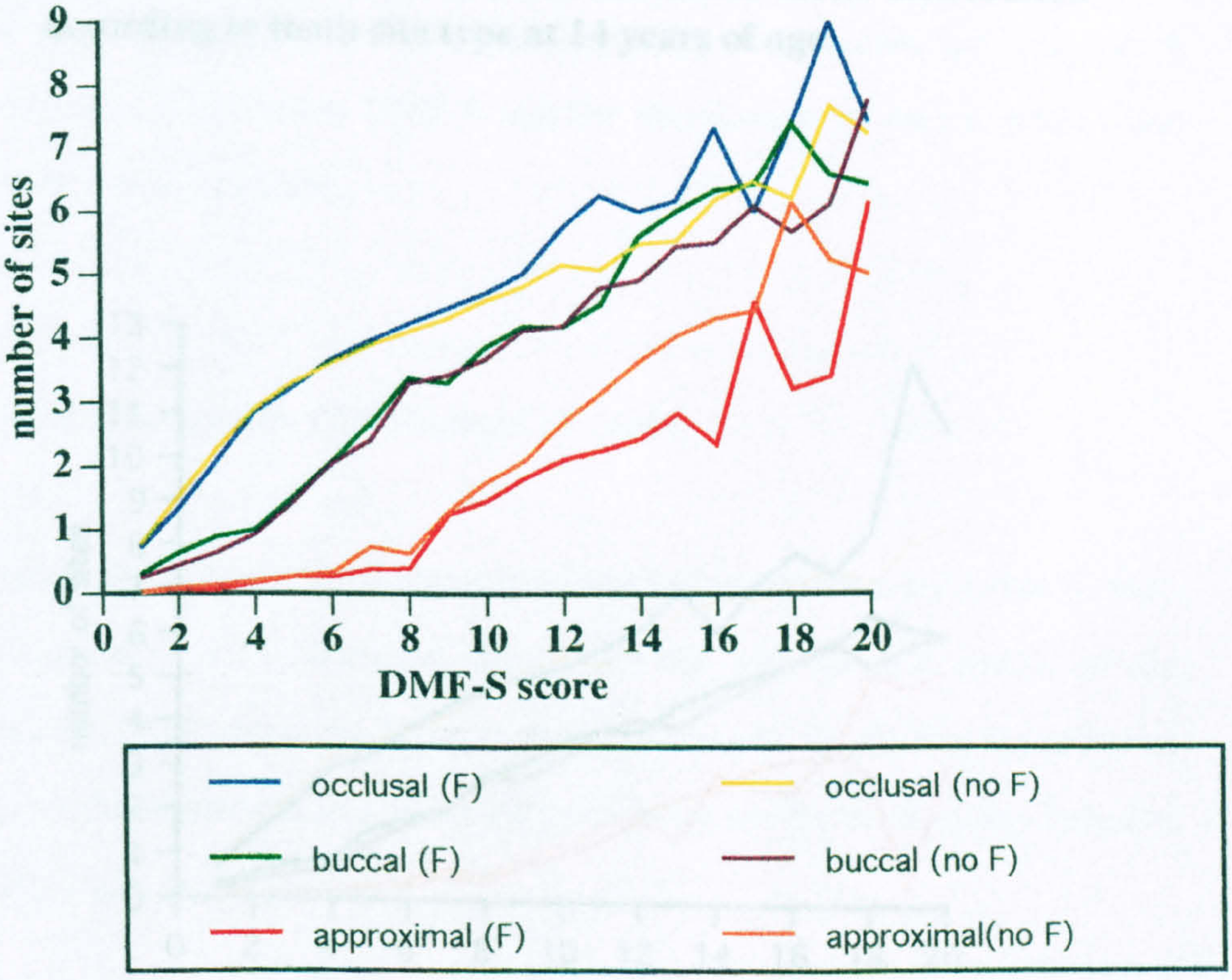


Figure 18: Effect of water fluoridation on caries distribution according to tooth site type at 12 years of age

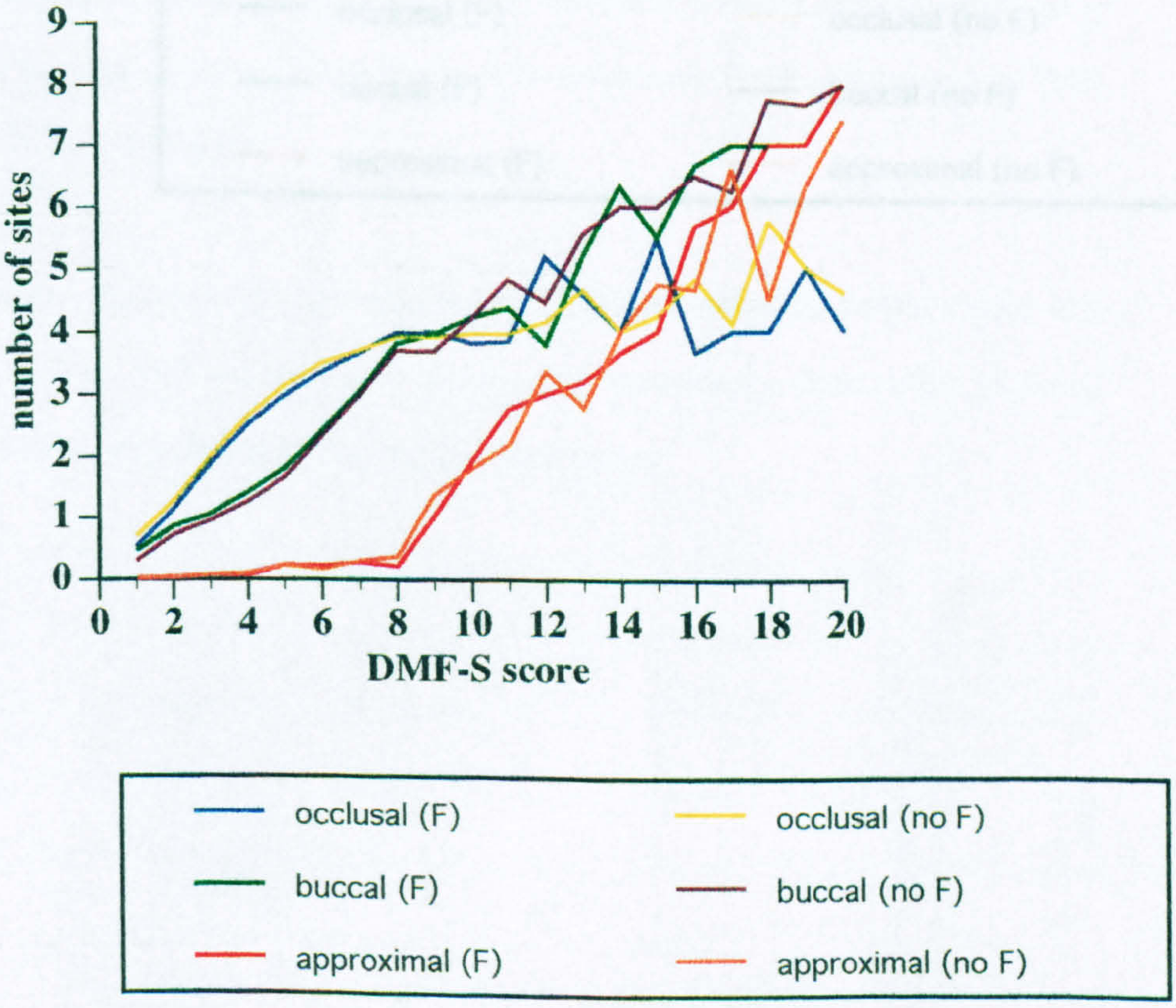
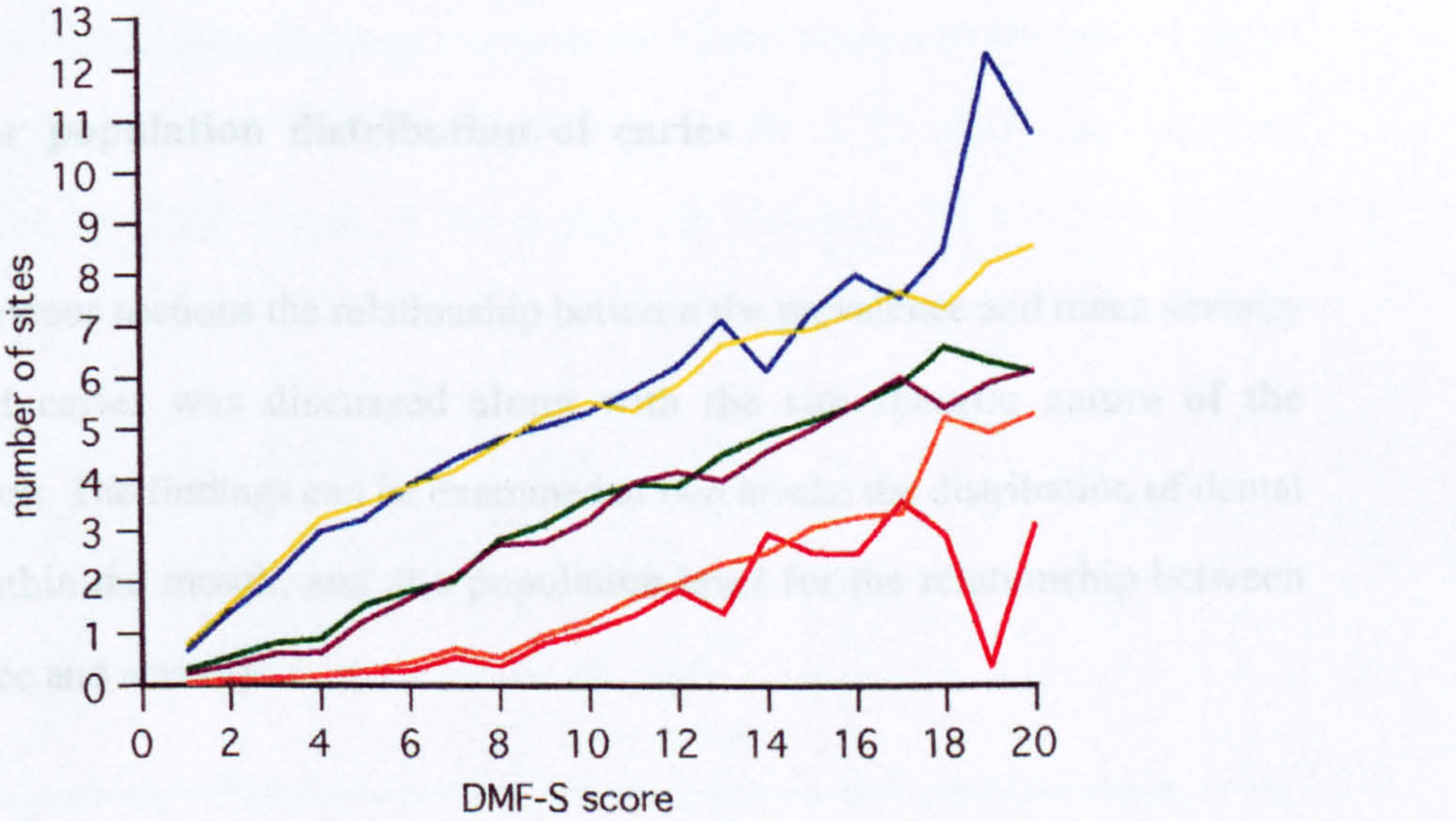


Figure 19: Effect of water fluoridation on caries distribution according to tooth site type at 14 years of age



If the distribution within a population can be ascertained, the number of individuals at a particular DMF-S and the distribution of caries within each individual can be predicted.

4.5: The inter population distribution of caries

In the previous sections the relationship between the prevalence and mean severity of dental caries was discussed along with the site specific nature of the distribution. The findings can be examined at two levels; the distribution of dental caries within the mouth, and at a population level for the relationship between prevalence and severity.

Within the mouth, certain sites were more prone to decay than others. The propensities was independent of fluoride status, sex, or ethnic background of the study population and at a population level, there was a mathematical relationship between the percentage of a population who were caries free and the mean DMF-S score of the population.

This section explores the epidemiological characteristics of dental caries at a population level and will deal with relationship between the distribution of dental caries within a population at varying levels of caries.

4.5.1: The relationship between the mean DMF-S and -T score and inter-population distribution

The data have been grouped according to the differing age groups and site locations. For each of the examinations over the study period the mean DMF-S and -T scores for each sub-group has been calculated along with the percentage of the sub-group at each DMF-S or -T. Figures 20 to 25 show the changing distribution of DMF-S and -T for 6 year-old children. Table 12 shows the changing distribution of DMF-S for mean DMF-S scores of 0.26, 0.34 and 0.62.

Table 12: Percentage distribution of population for mean DMF-S scores of 0.26 to 0.62 at 6 years of age.

mean	individual DMF-S score								
DMF-S	0	1	2	3	4	5	6	7	8
0.26	90	3	2	2	1				
0.34	85	7	3	2	1	1	1	1	
0.62	74	20	6	3	3	3			

As the mean DMF-S score increases the percentage of caries free individuals falls and there is a graduated increase in the spread of those individuals with at least one surface experiencing caries, (Figures 20 to 22). As reported in section 4.2, the change in mean DMF-S scores is not due to a separate group getting more caries. The percentage with no caries experience declines as the mean DMF-S score increases. This pattern of increase in the percentage is repeated at DMF-S scores of between 1 and 3. As the spread of the distribution increases, there is a

Figure 20: Distribution of caries for 6 year-old populations with mean DMF-S scores of 0.26.

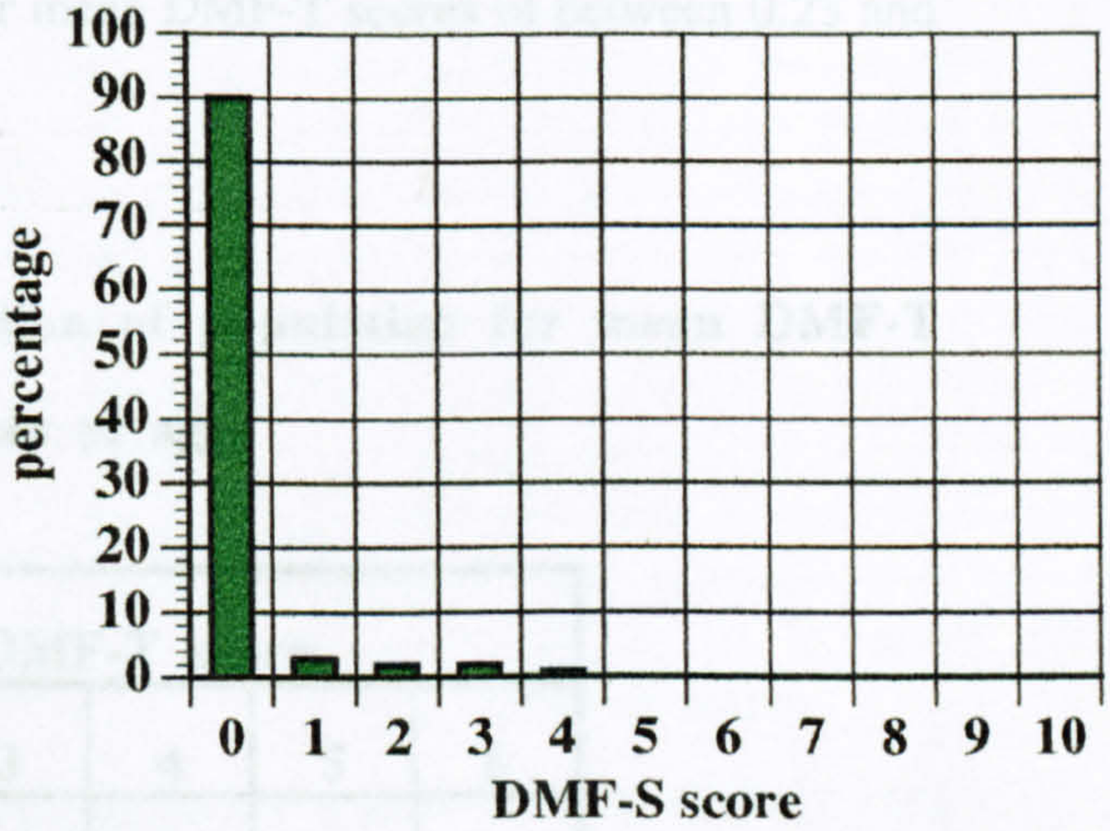


Figure 21: Distribution of caries for 6 year-old populations with mean DMF-S scores of 0.34.

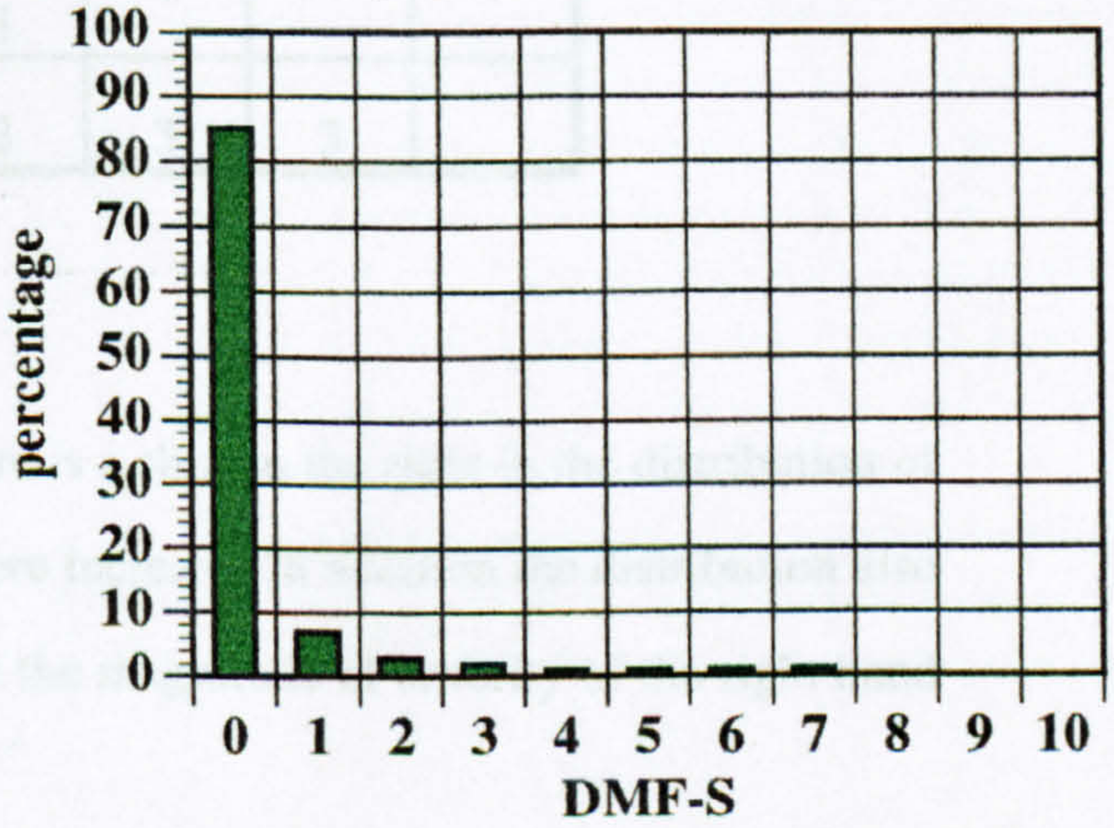
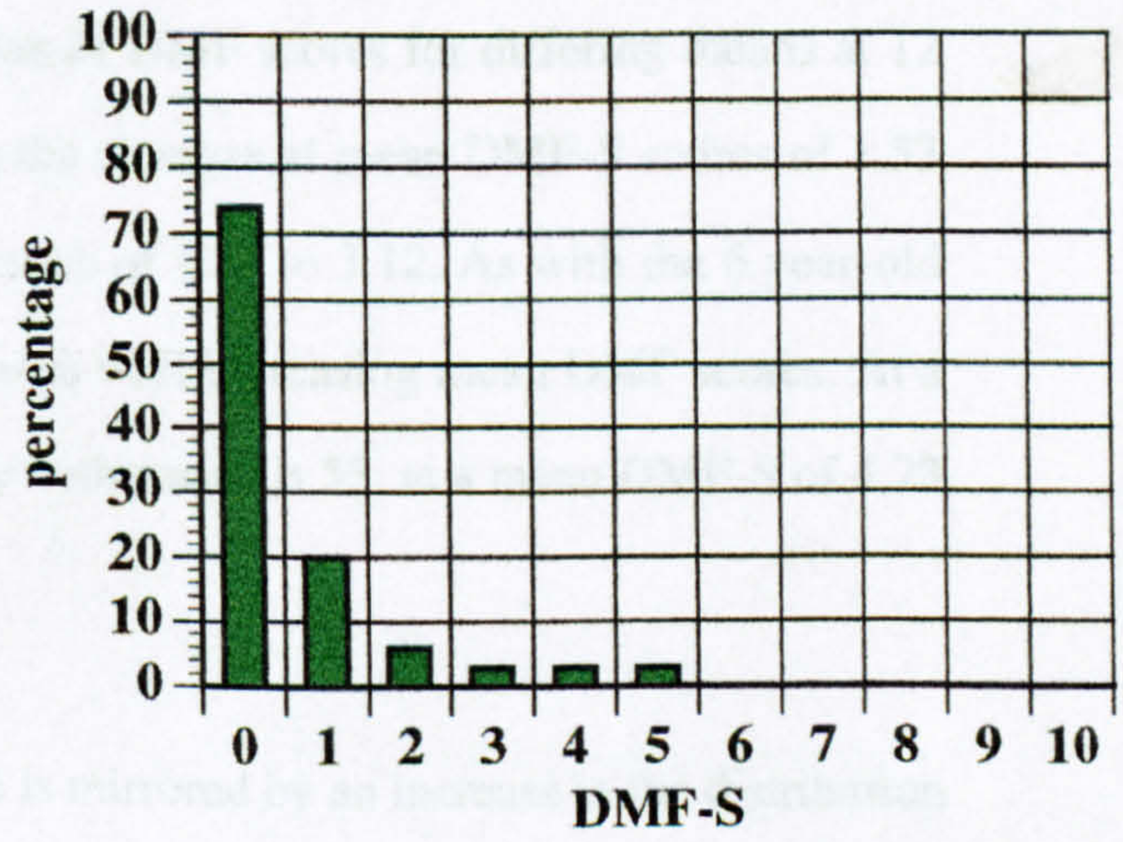


Figure 22: Distribution of caries for 6 year-old populations with mean DMF-S scores of 0.62.



population shift. Table 13 and Figures 23 to 25 shows the changing distribution for DMF-T scores in 6 year-olds for mean DMF-T scores of between 0.23 and 0.57.

Table 13: Percentage distribution of population for mean DMF-T scores of 0.23 to 0.57 at 6 years of age.

mean	individual DMF-T score						
DMF-T	0	1	2	3	4	5	6
0.23	90	3	2	2	1		
0.39	86	8	5	1			
0.57	70	13	10	3	3	3	

As with the DMF-S distribution there is a shift to the right in the distribution of the severity as the mean DMF-T score increase. In addition the distribution also widens; there is a steady increase in the magnitude of severity of the right hand side of the population distribution.

Figures 26 to 31 show the distribution of DMF scores for differing means at 12 years of age. Figures 26 to 28 show the changes at mean DMF-S scores of 1.53 to 4.73, Figures 29 to 31 DMF-T scores of 1.27 to 3.12. As with the 6 year-old data, the percentage caries free declines with increasing mean DMF scores. At a mean DMF-S of 1.53 the percentage with caries is 53, at a mean DMF-S of 4.73 this had dropped to 23%.

The decline in percentage caries free is mirrored by an increase in the distribution of caries within the population. For example 5% of individuals are found with a DMF-S score of 4 when the mean DMF-S of the population is 1.53 whilst when

Figure 23: Distribution of caries for 6 year-old populations with mean DMF-T scores of 0.23.

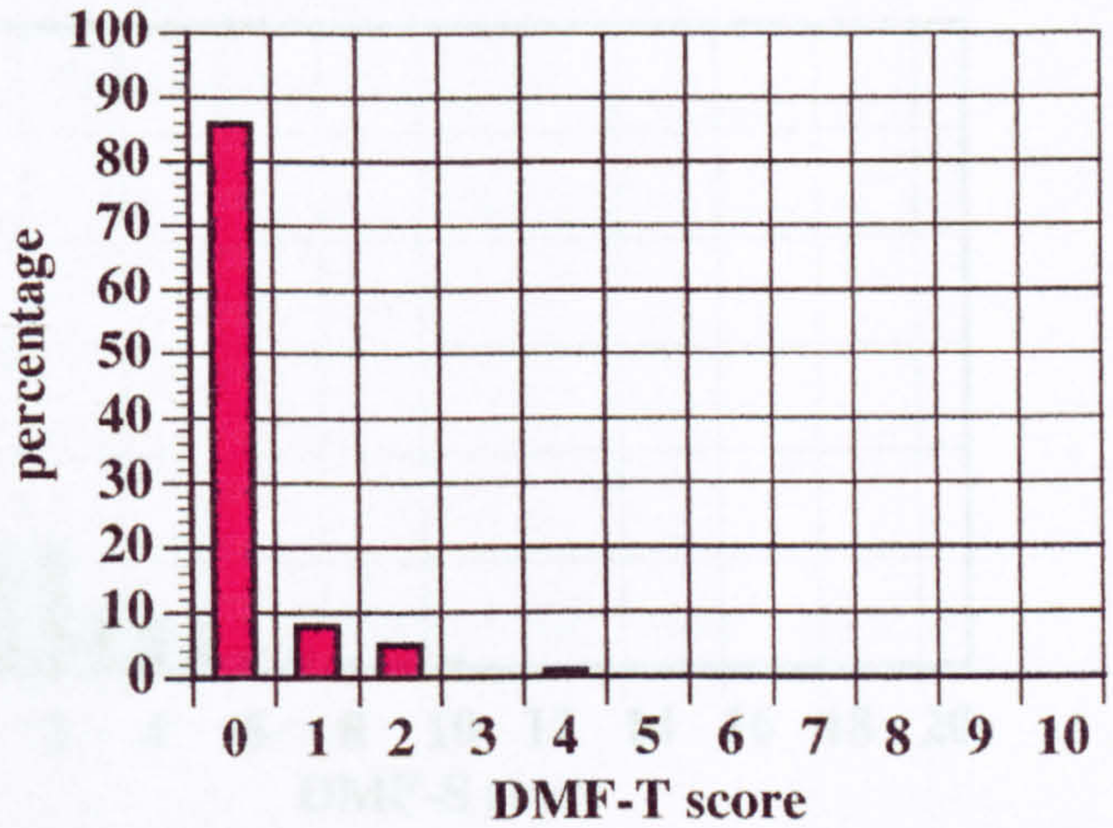


Figure 24: Distribution of caries for 6 year-old populations with mean DMF-T scores of 0.39.

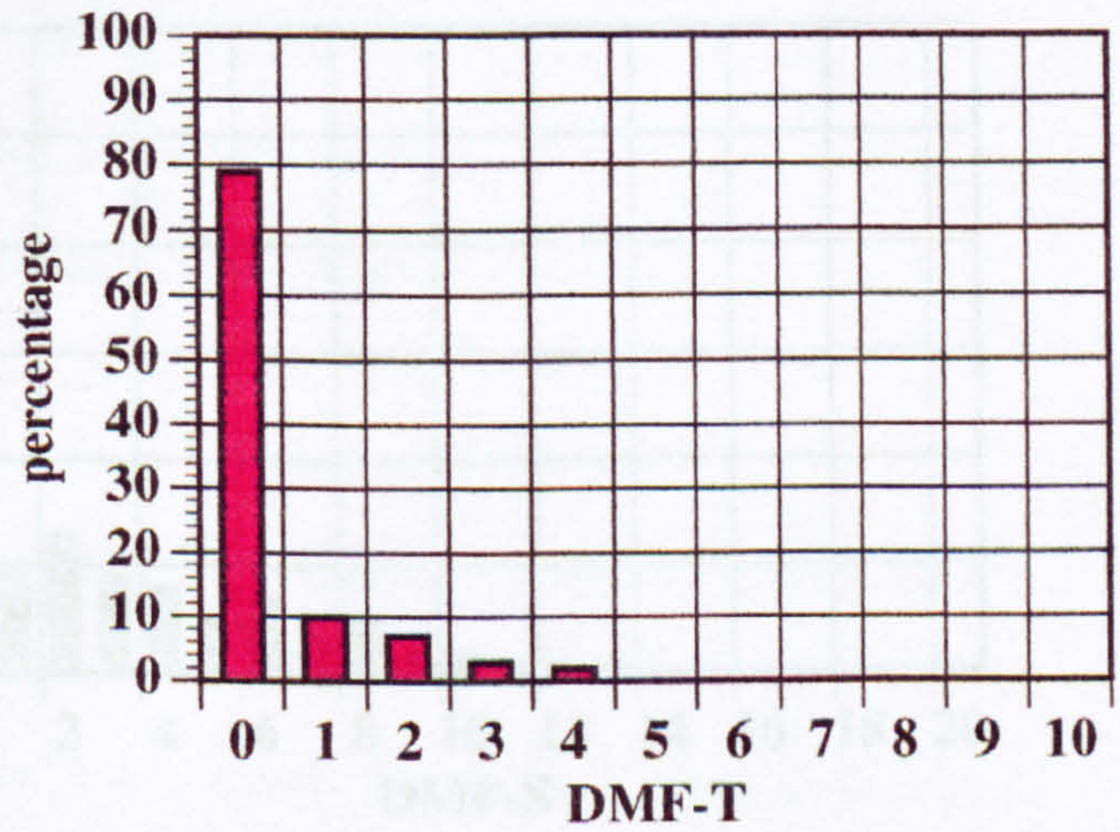


Figure 25: Distribution of caries for 6 year-old populations with mean DMF-T scores of 0.57.

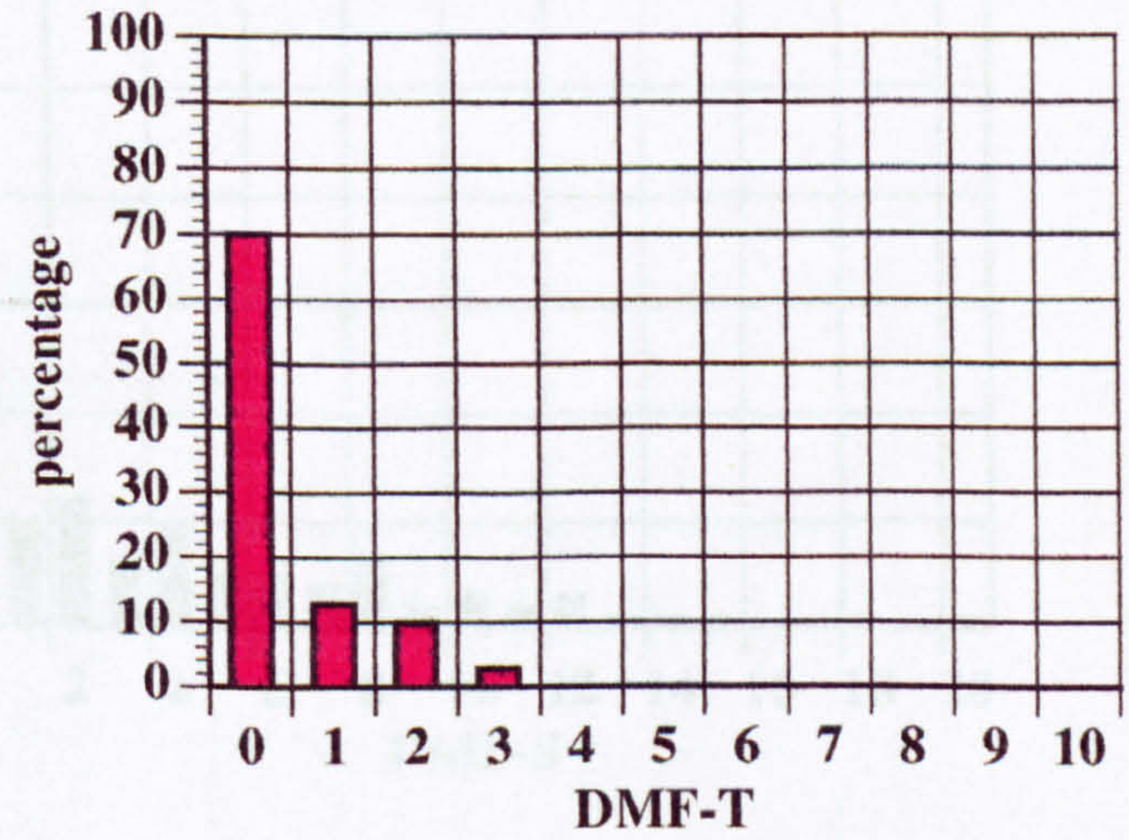


Figure 26: Distribution of caries for 12 year-old populations with mean DMF-S scores of 1.53

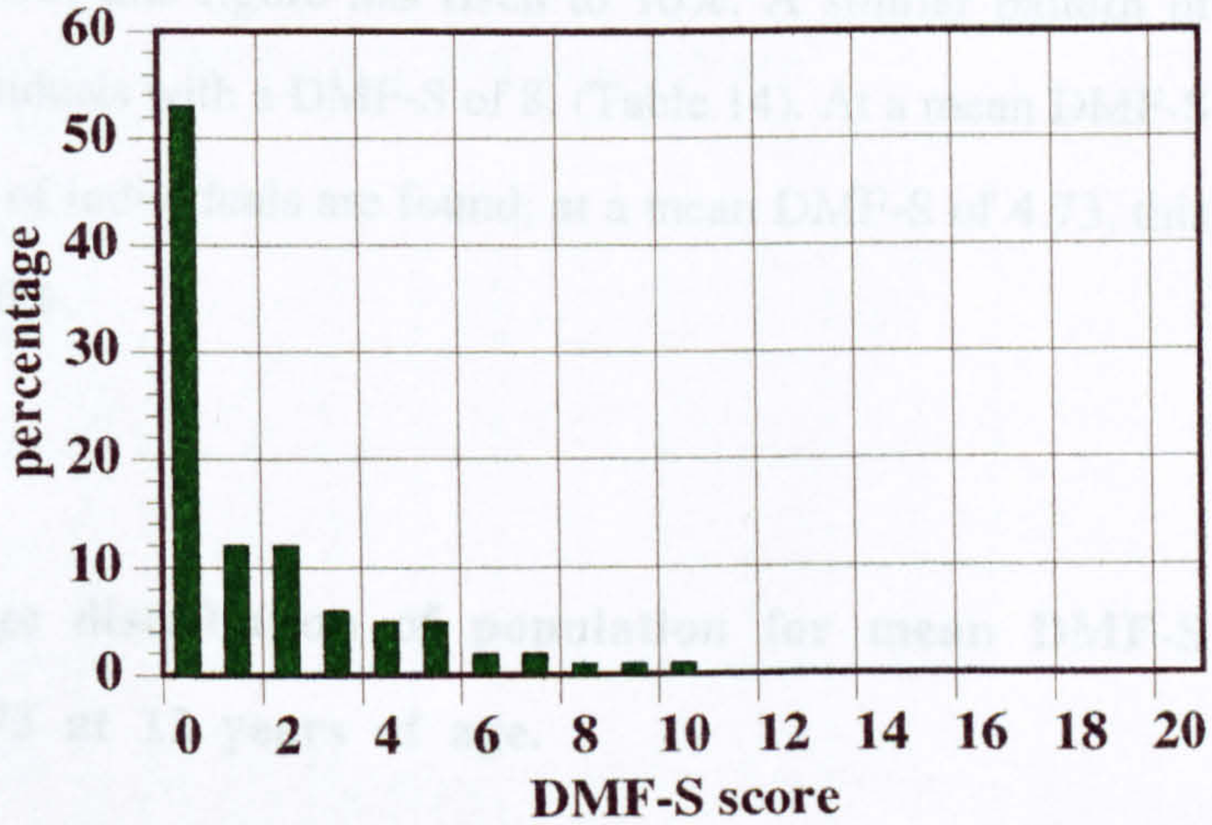


Figure 27: Distribution of caries for 12 year-old populations with mean DMF-S scores of 3.36

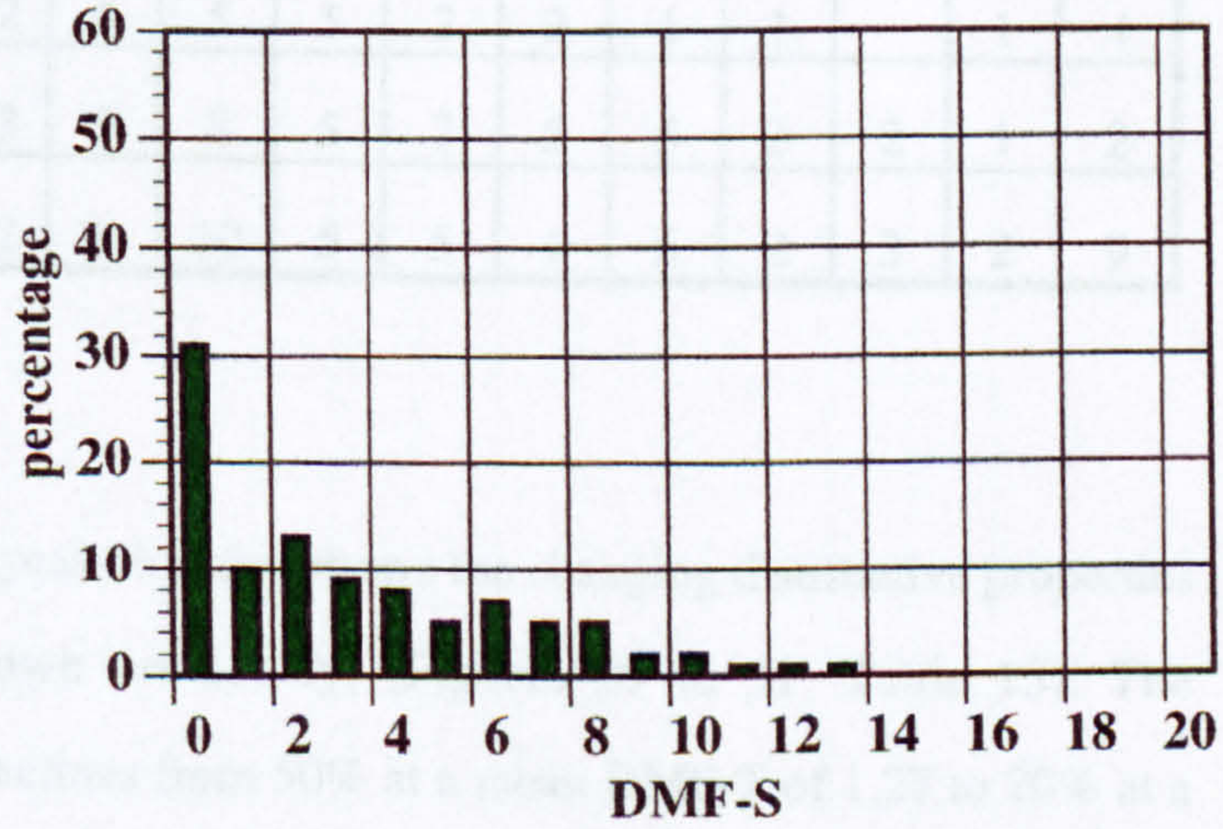
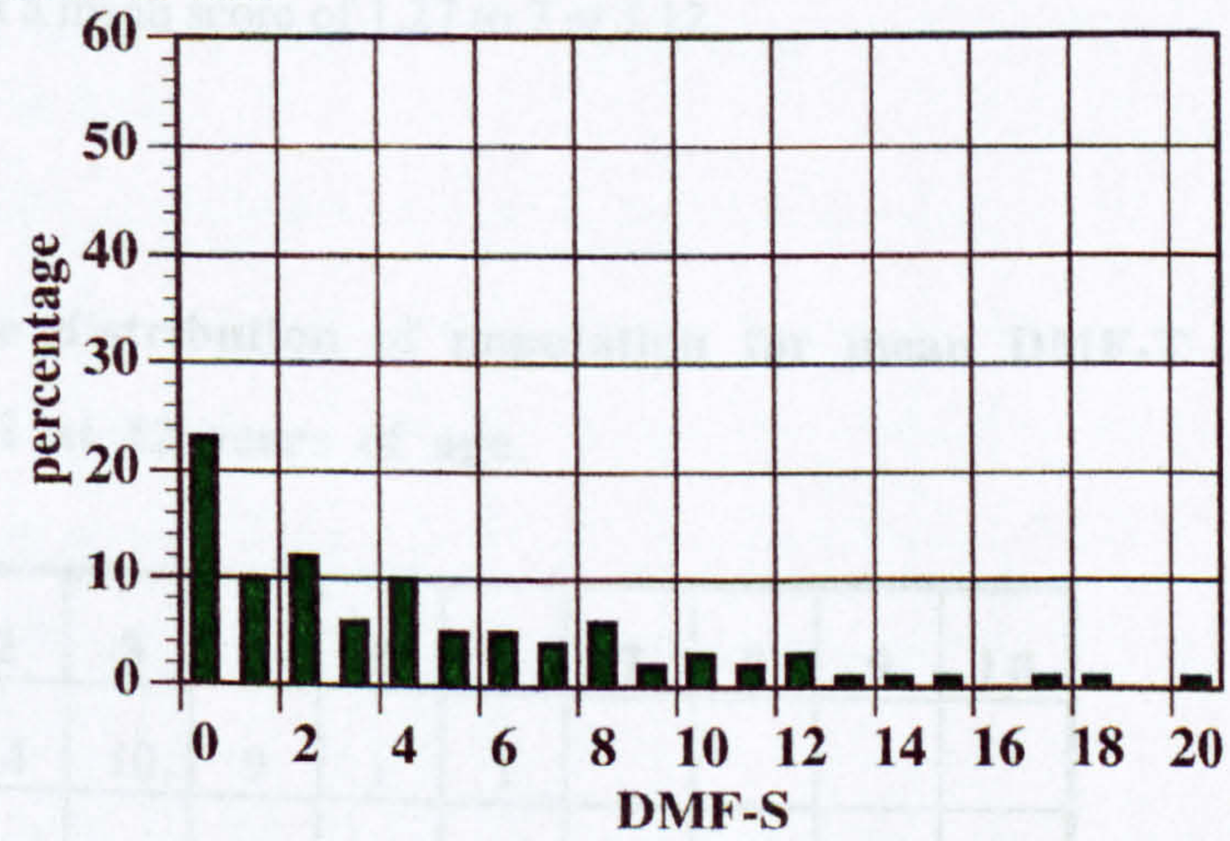


Figure 28: Distribution of caries for 6 year-old populations with mean DMF-S scores of 4.73



the mean DMF-S is 4.73, this figure has risen to 10%. A similar pattern of increase is seen in individuals with a DMF-S of 8, (Table 14). At a mean DMF-S score of 1.53, only 1% of individuals are found; at a mean DMF-S of 4.73, this figure had increased to 6%.

Table 14: Percentage distribution of population for mean DMF-S scores of 1.53 to 4.73 at 12 years of age.

DMF-S	0	1	2	3	4	5	6	7	8	9	10	11	+12
1.53	53	12	12	6	5	5	2	2	1	1		1	4
3.36	31	10	13	9	8	5	7	5	5	2	2	1	2
4.73	23	10	12	6	10	5	5	4	6	2	3	2	9

The DMF-T data for 12 year-olds also shows the changing distributive properties seen in DMF data shown previously, (Figures 29 to 31; Table 15). The percentage caries free declines from 50% at a mean DMF-T of 1.27 to 20% at a DMF-T of 3.12. The increase in the percentage of individuals with a DMF-T score of 5 rises from 1 at a mean score of 1.27 to 7 at 3.12.

Table 15: Percentage distribution of population for mean DMF-T scores of 1.27 to 3.12 at 12 years of age.

DMF-T	0	1	2	3	4	5	6	7	8	9	10
1.27	50	14	14	10	9	1	1				
2.21	30	11	15	12	23	5	3	1			1
3.12	20	11	16	11	22	7	3	2	3	3	1

Figure 29: Distribution of caries for 12 year-old populations with mean DMF-T scores of 1.27

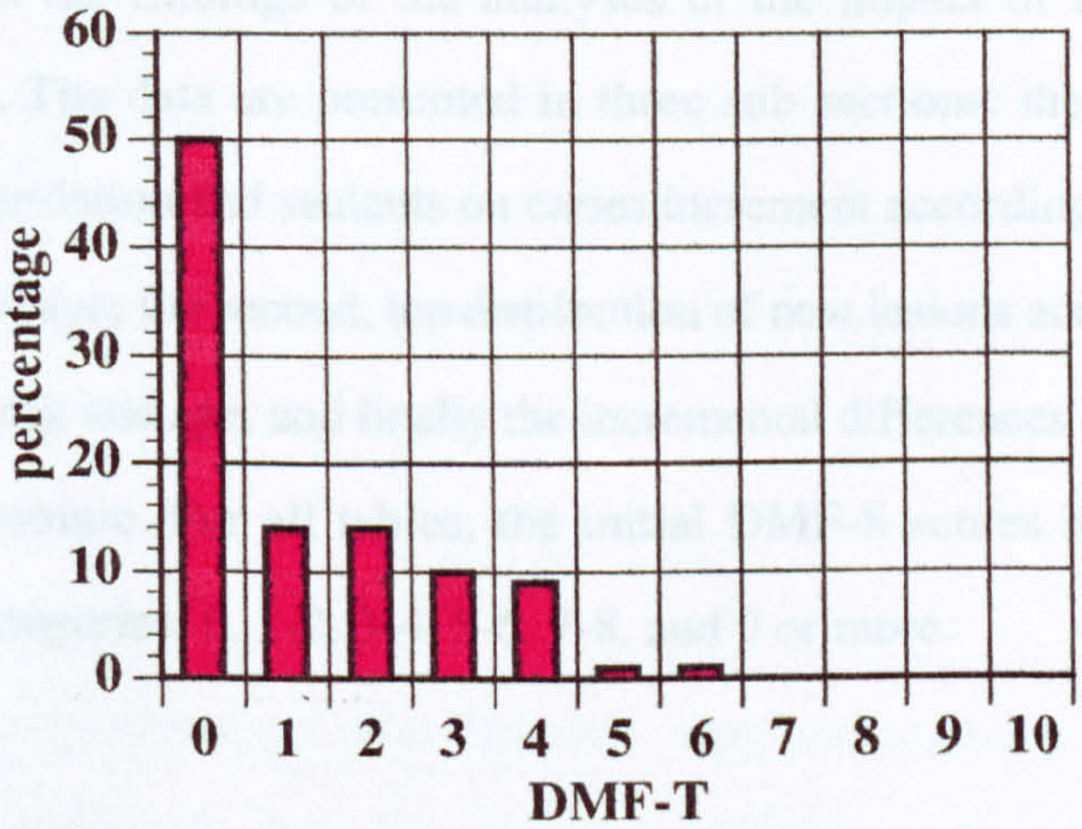


Figure 30: Distribution of caries for 12 year-old populations with mean DMF-T scores of 2.21

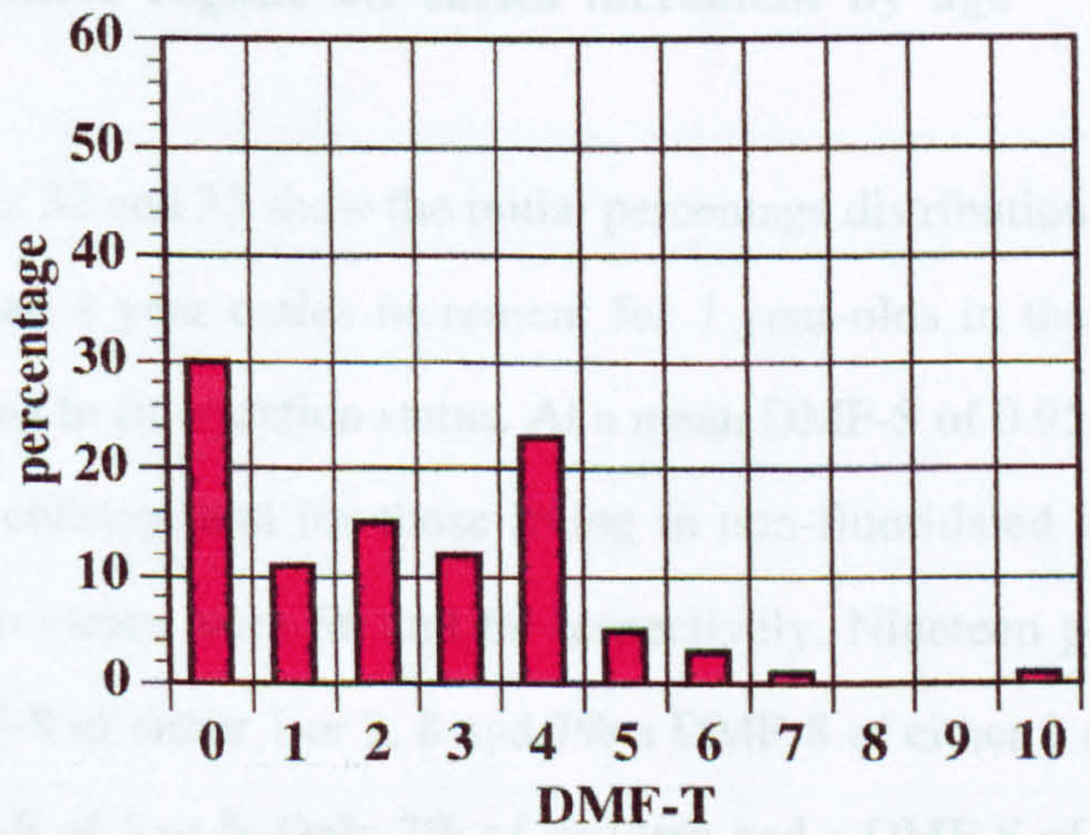
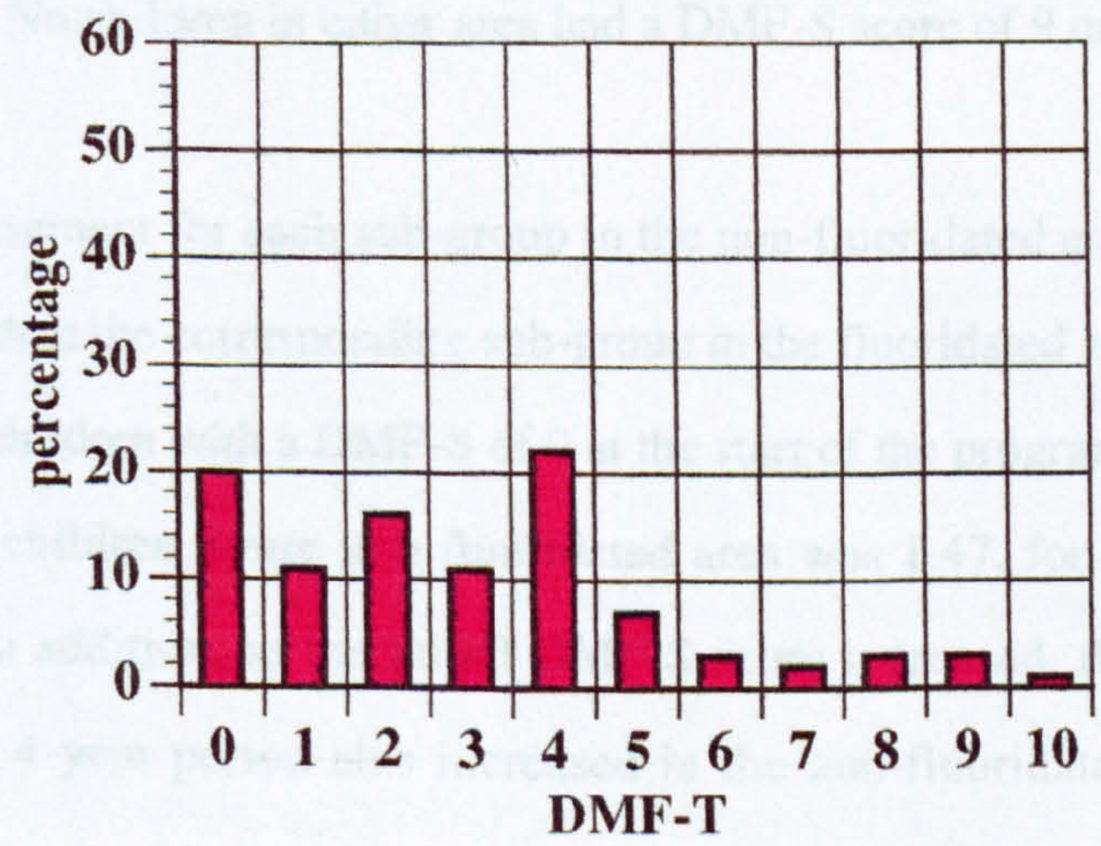


Figure 31: Distribution of caries for 12 year-old populations with mean DMF-T scores of 3.12



4.6: Impact of preventive regime on caries increments

This section reports the findings of the analyses of the impact of the caries preventive regimes. The data are presented in three sub sections: the first, the impact of water fluoridation and sealants on caries increment according to initial severity of attack and age; the second, the distribution of new lesions according to initial severity of attack and age, and finally the incremental differences according to preventive programme. For all tables, the initial DMF-S scores have been grouped into five categories; 0, 1-2, 3-4, 5-6, 7-8, and 9 or more.

4.6.1: Impact of preventive regime on caries increment by age

Table 16 and Figures 32 and 33 show the initial percentage distribution of caries and subsequent mean 4 year caries increment for 7 year-olds in the NPDDP programme according to fluoridation status. At a mean DMF-S of 0.95 and 0.82 for the fluoridated children and for those living in non-fluoridated areas, the percentages with no caries were 66 and 69 respectively. Nineteen percent of children had a DMF-S of either 1 or 2, 8 and 7% a DMF-S of either 3 or 4 , and 4% and 3% a DMF-S of 5 or 6. Only 2% of children had a DMF-S of 7 or 8 in the fluoridated area, although this was a higher figure than the non-fluoridated areas with only 1%. No children in either area had a DMF-S score of 9 or more.

The mean caries increment for each sub-group in the non-fluoridated areas were consistently higher than the corresponding sub-group in the fluoridated areas. For example, for those children with a DMF-S of 0 at the start of the programme, the increment in those children living in a fluoridated area was 1.47, for the non-fluoridated, 1.88. In addition, as the initial DMF-S score increased, the caries increment over the 4 year period also increased in the non-fluoridated areas,

although the mean increment for those children with a DMF-S of 7 or 8 at the commencement of the programme was slightly lower than those with a DMF-S of 5 or 6, 3.56 against 3.78. For those children living in a fluoridated area, the increments did not show a similar pattern. Although for those children with a DMF-S of 1 or 2 and 3 or 4, the increment was higher when compared to the children with a DMF-S score of 0, for children with an initial DMF-S of 5 or higher, the increments were lower.

Table 16: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 7 year-old children according to fluoridation status.

	Non Fluoridated		Fluoridated	
DMF-S Group	percentage	increment	percentage	increment
0	66	1.88	69	1.47
1-2	19	2.90	19	2.05
3-4	8	3.26	7	2.06
5-6	4	3.84	3	1.43
7-8	2	3.56	1	1.24
9 or more	-	-	-	-

A similar pattern existed for the 9 year-olds, Table 17 and Figures 34 and 35. The increments for the group resident in the non-fluoridated areas were higher than for those in the fluoridated areas, most markedly for the high initial caries groups. As previously, there was an increase in the mean 4 year increment according to initial caries levels in the non-fluoridated group, rising from 1.84 for those individuals with a DMF-S score of 0 at the programme commencement, to 10.35 for those

Figure 32: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 7 year-old children in non-fluoridated areas

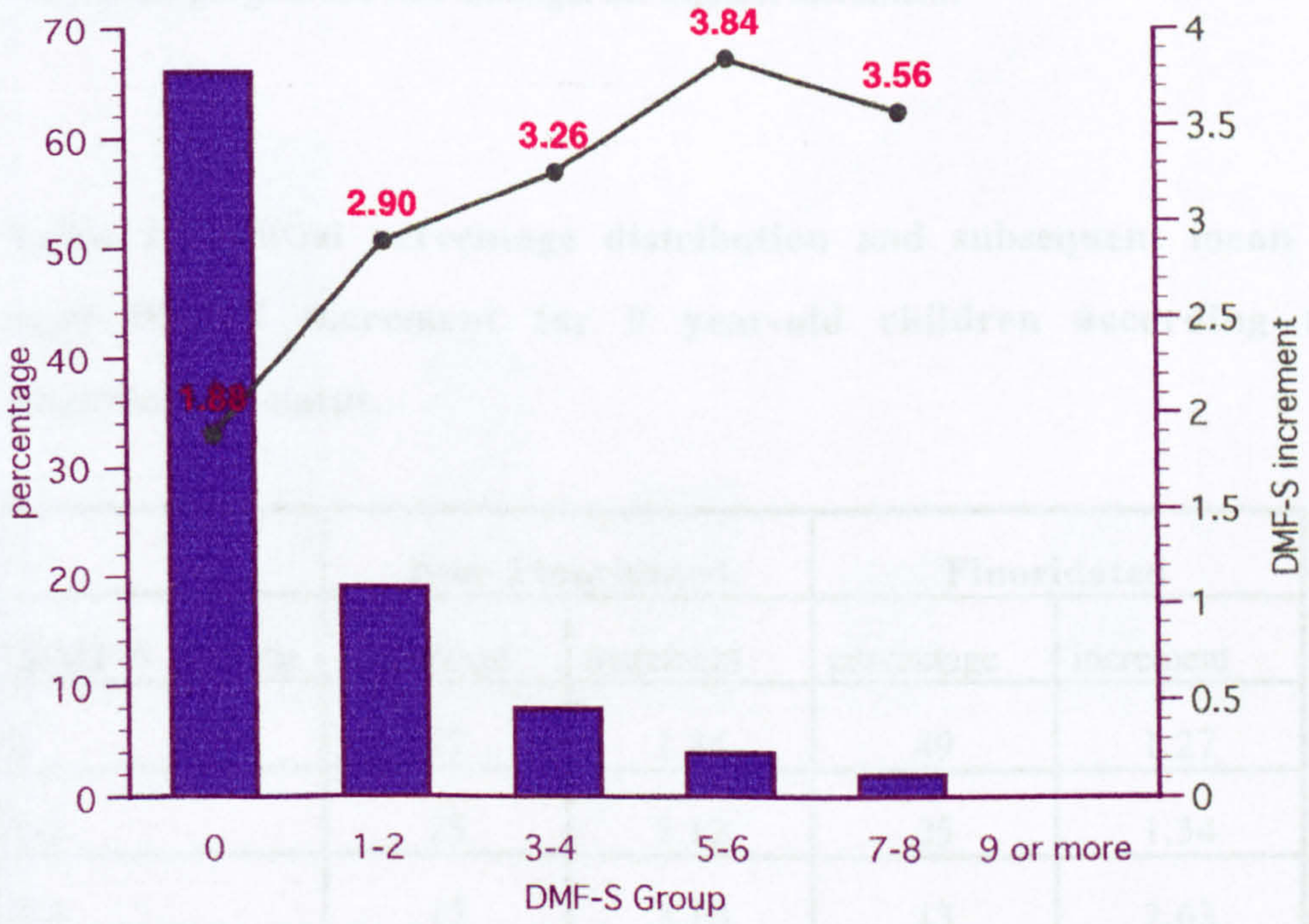
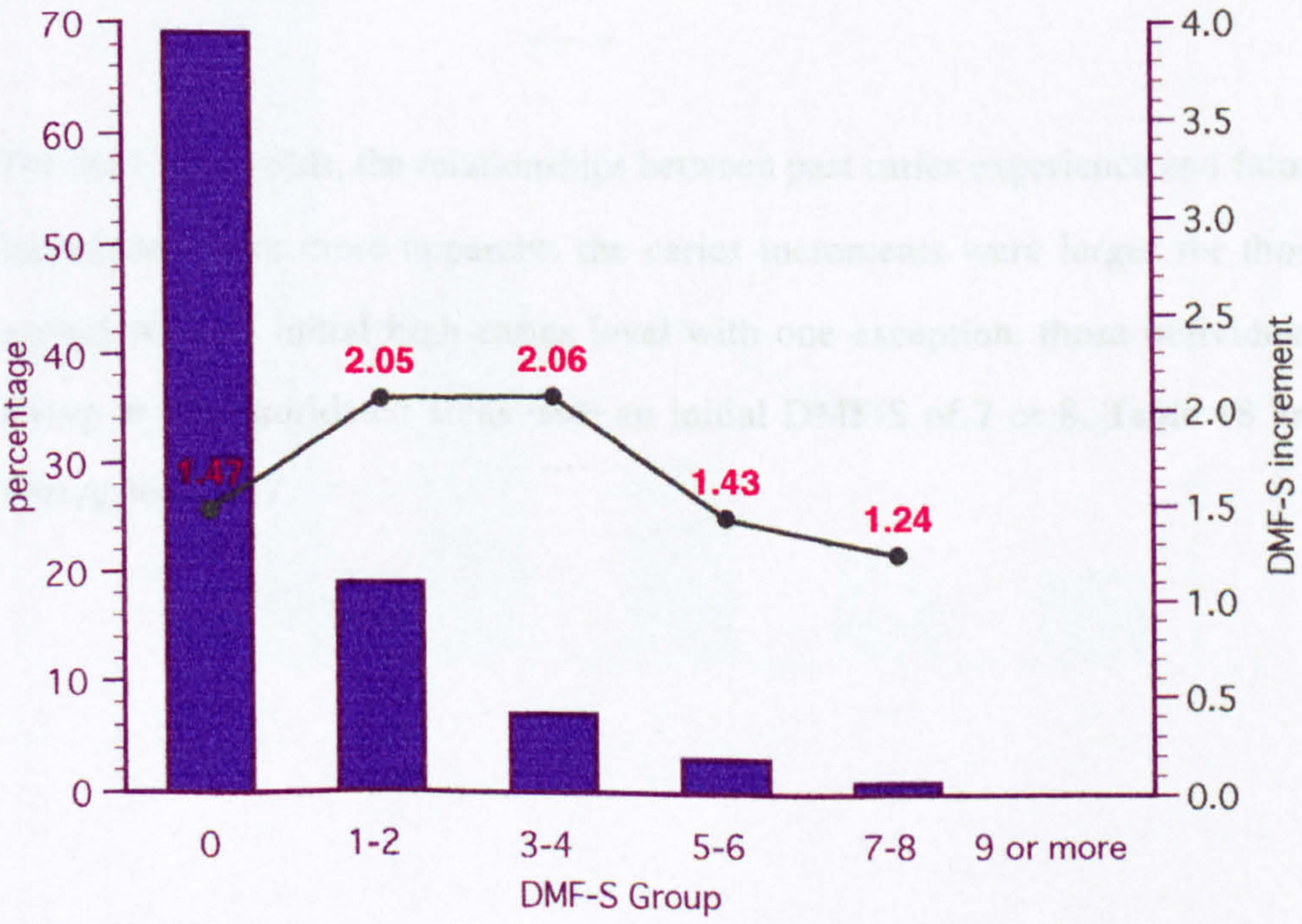


Figure 33: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 7 year-old children in fluoridated areas



with a DMF-S of 9 or more at the start. For the fluoridated groups, the increments were consistently lower than for the non-fluoride groups, and, as with the 7 year-olds living in fluoridated areas, those individuals with a DMF-S or 3 or 4 at the start of the programme had amongst the highest increment.

Table 17: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 9 year-old children according to fluoridation status.

	Non Fluoridated		Fluoridated	
DMF-S Group	percentage	increment	percentage	increment
0	37	1.84	49	1.27
1-2	25	3.12	25	1.34
3-4	15	3.10	13	2.63
5-6	10	4.48	7	1.86
7-8	9	2.96	5	1.71
9 or more	4	10.35	1	2.67

For the 11 year-olds, the relationships between past caries experience and future increments were more apparent: the caries increments were larger for those groups with an initial high caries level with one exception, those individuals living in the fluoridated areas with an initial DMF-S of 7 or 8, Table 18 and Figure 36 and 37.

Figure 34: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 9 year-old children in non-fluoridated areas

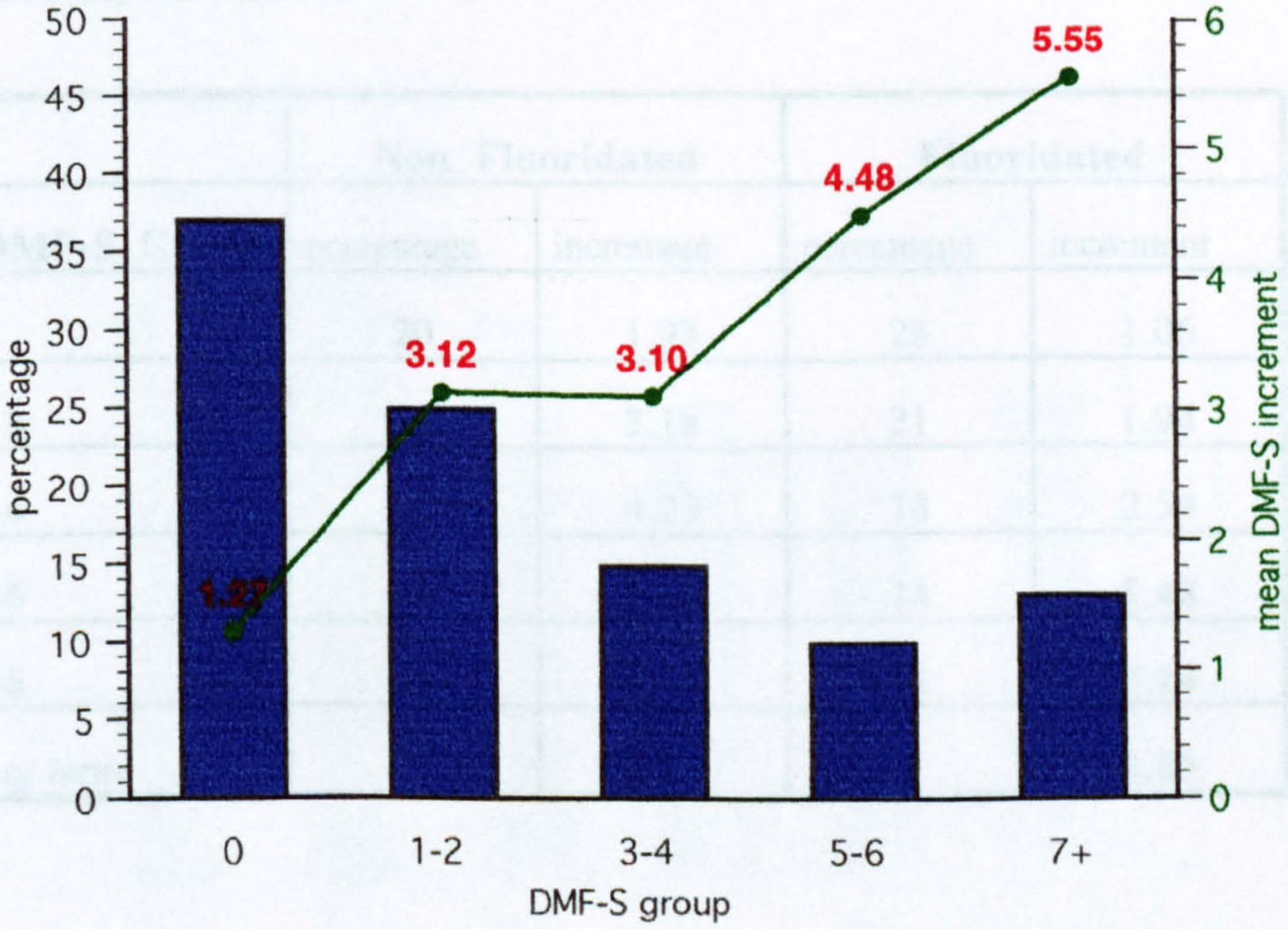


Figure 35: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 9 year-old children in fluoridated areas

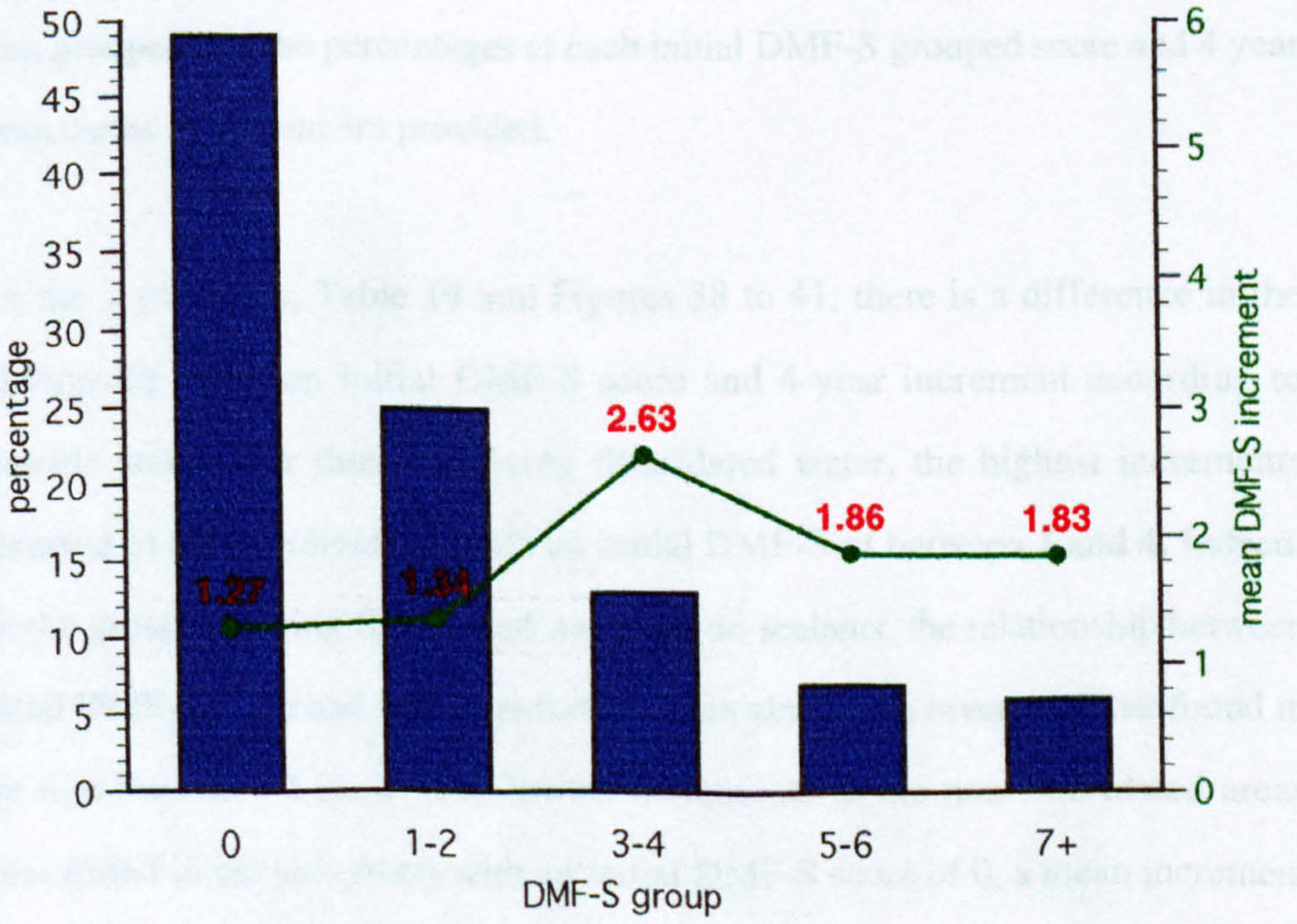


Table 18: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 11 year-old children according to fluoridation status.

	Non Fluoridated		Fluoridated	
DMF-S Group	percentage	increment	percentage	increment
0	20	1.93	28	1.06
1-2	18	3.18	21	1.94
3-4	18	4.23	18	2.54
5-6	15	4.58	14	3.48
7-8	13	4.65	11	3.14
9 or more	16	8.14	8	4.85

Tables 19 and 20 and Figures 38 to 45 show the results of the mean caries increment analysed by programme type subdivided into those receiving fissure sealants and those not. As previously, the DMF-S scores for individuals have been grouped and the percentages at each initial DMF-S grouped score and 4 year mean caries increment are provided.

For the 7 year-olds, Table 19 and Figures 38 to 41, there is a difference in the relationship between initial DMF-S score and 4 year increment according to fluoride status. For those receiving fluoridated water, the highest increments occurred in those individuals with an initial DMF-S of between 1 and 4. Indeed, for the group receiving fluoridated water but no sealants, the relationship between initial DMF-S score and future increments was almost the inverse of that found in the non-fluoridated areas. The lowest increments in the non-fluoridated areas were found in the sub-group with an initial DMF-S score of 0, a mean increment of 1.08 for those with sealants and 2.28 for those without sealants, in the

Figure 36: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 11 year-old children in non-fluoridated areas

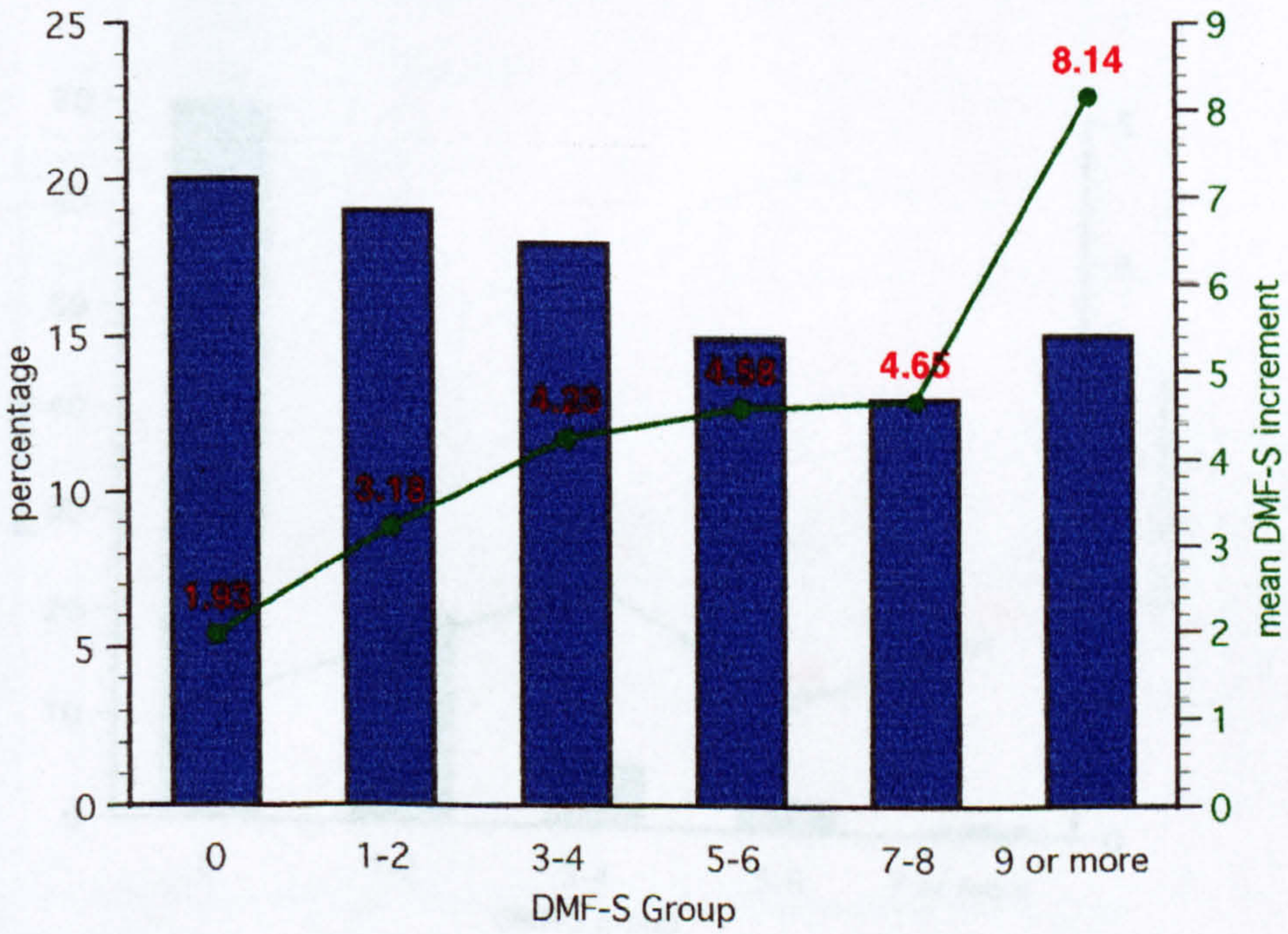


Figure 37: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 11 year-old children in fluoridated areas

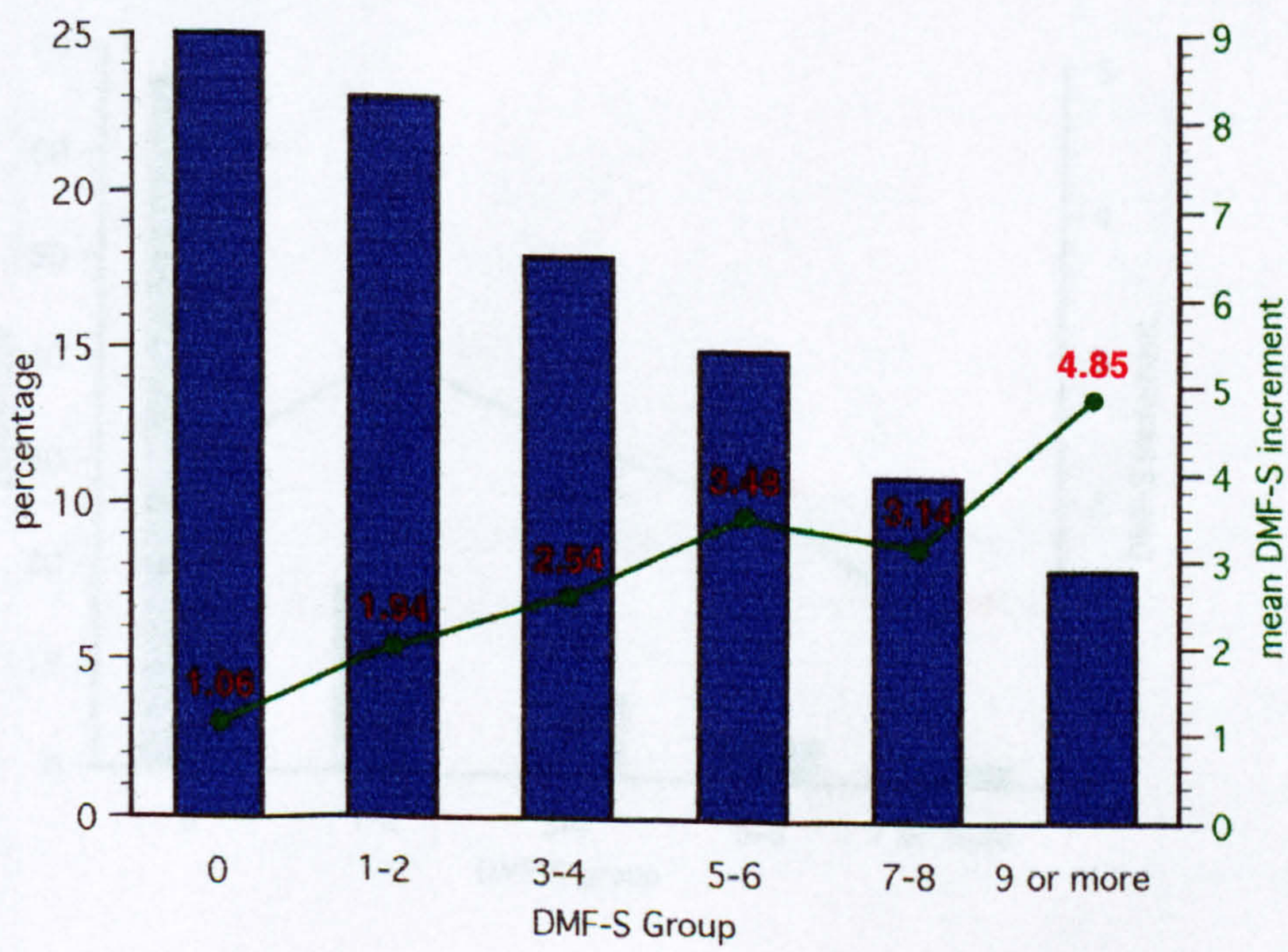


Figure 38: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 7 year-old children in fluoridated areas and receiving fissure sealants

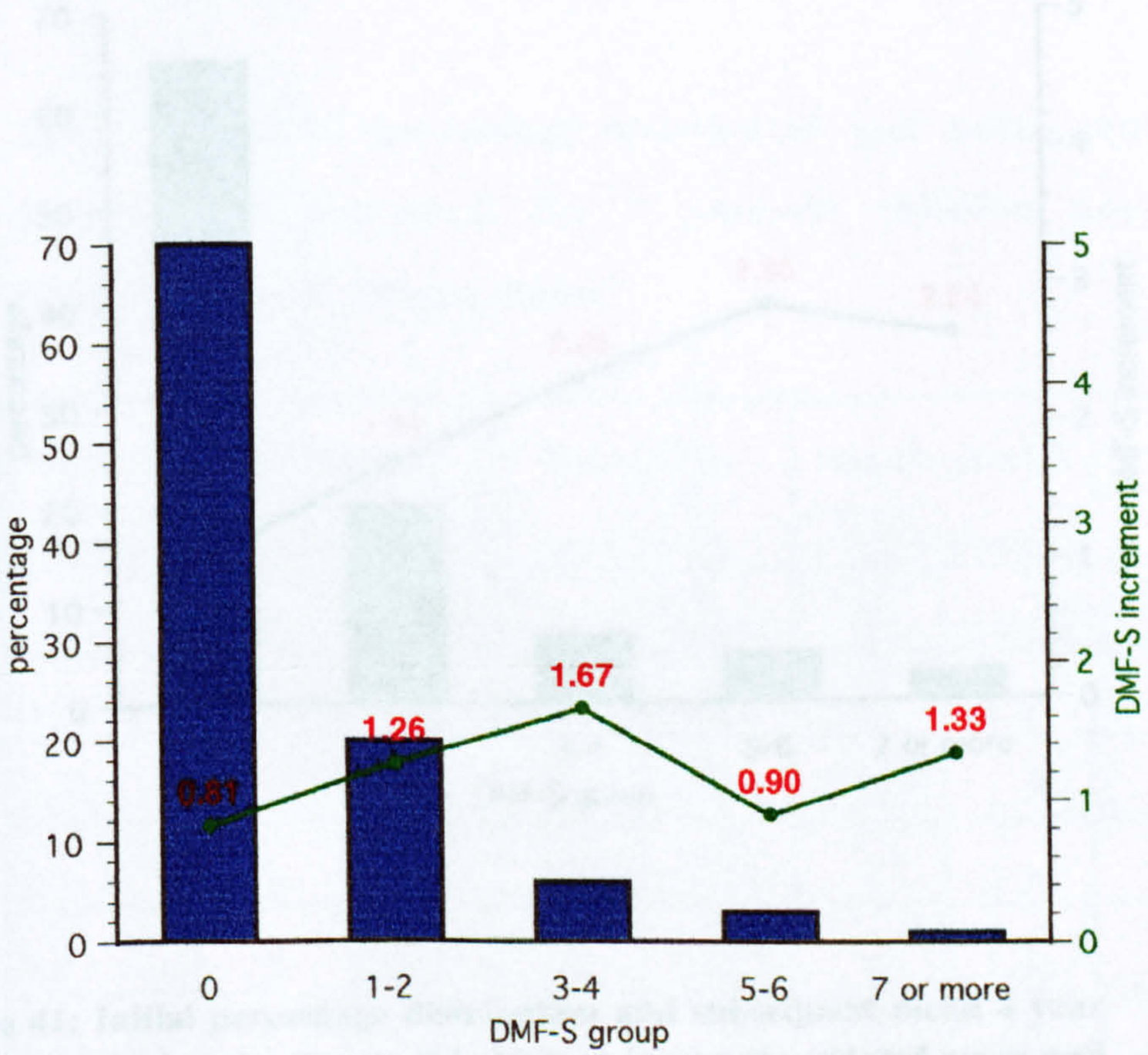


Figure 39: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 7 year-old children in fluoridated areas and not receiving fissure sealants

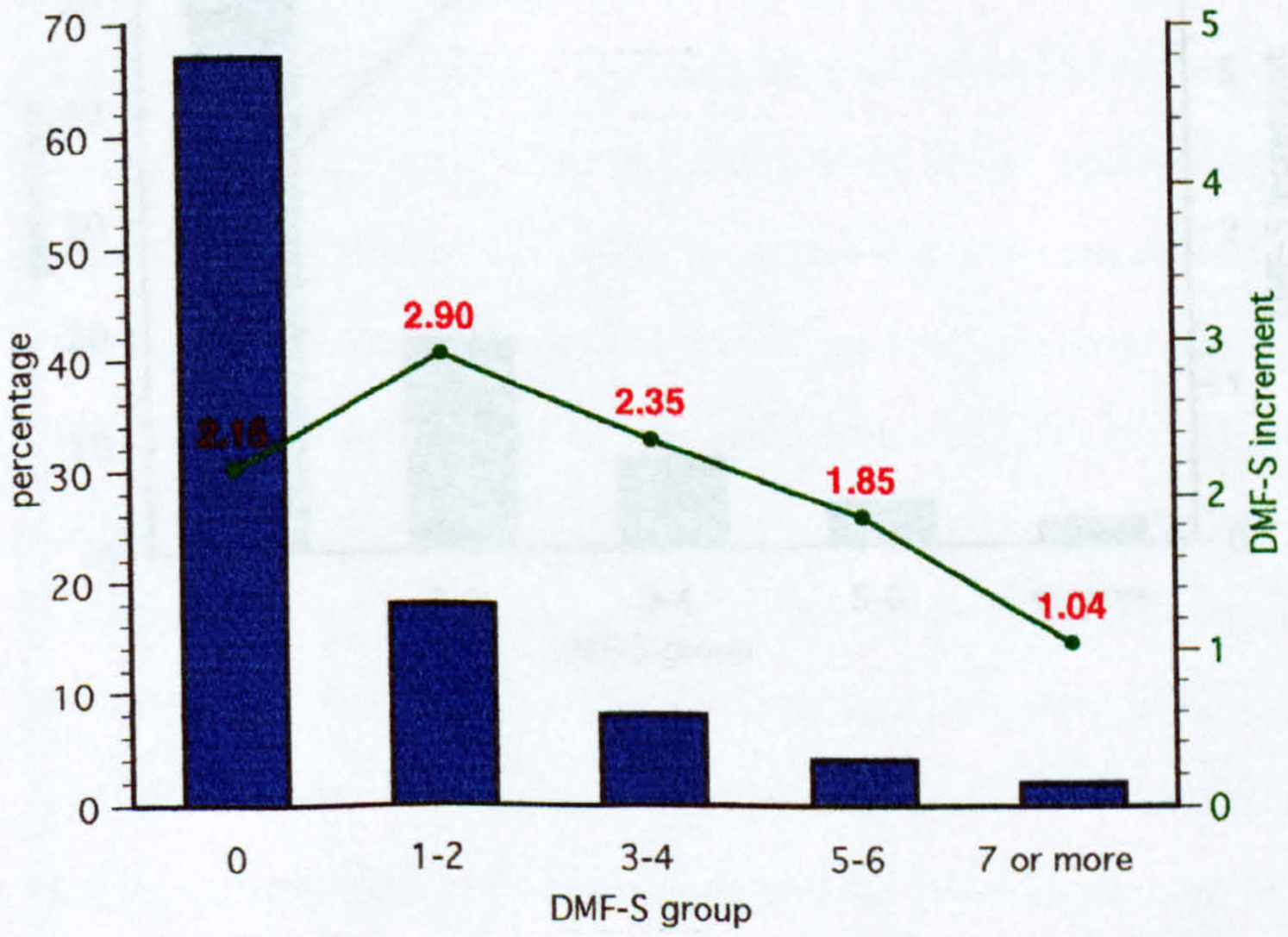


Figure 40: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 7 year-old children in non-fluoridated areas and receiving fissure sealants

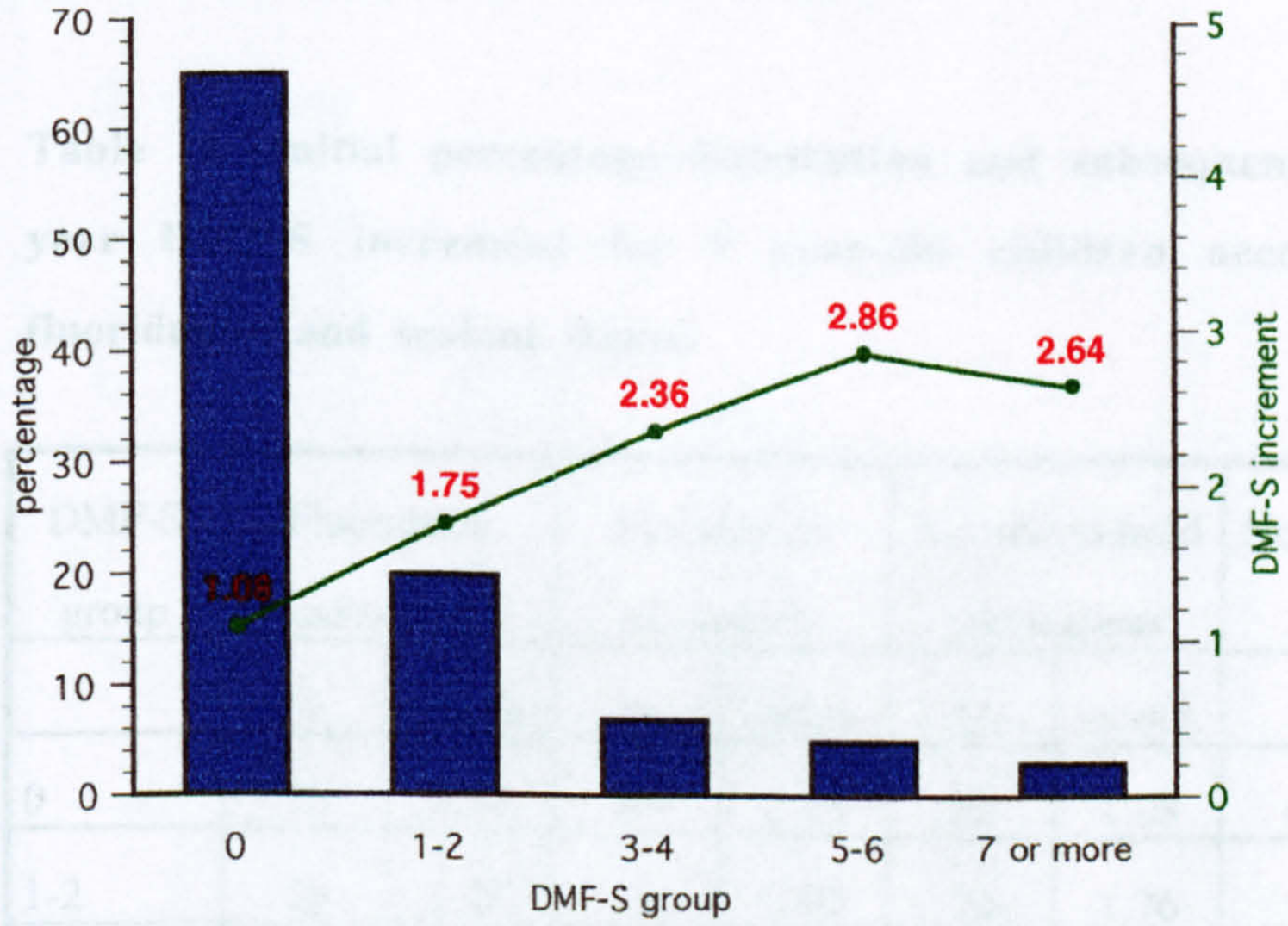
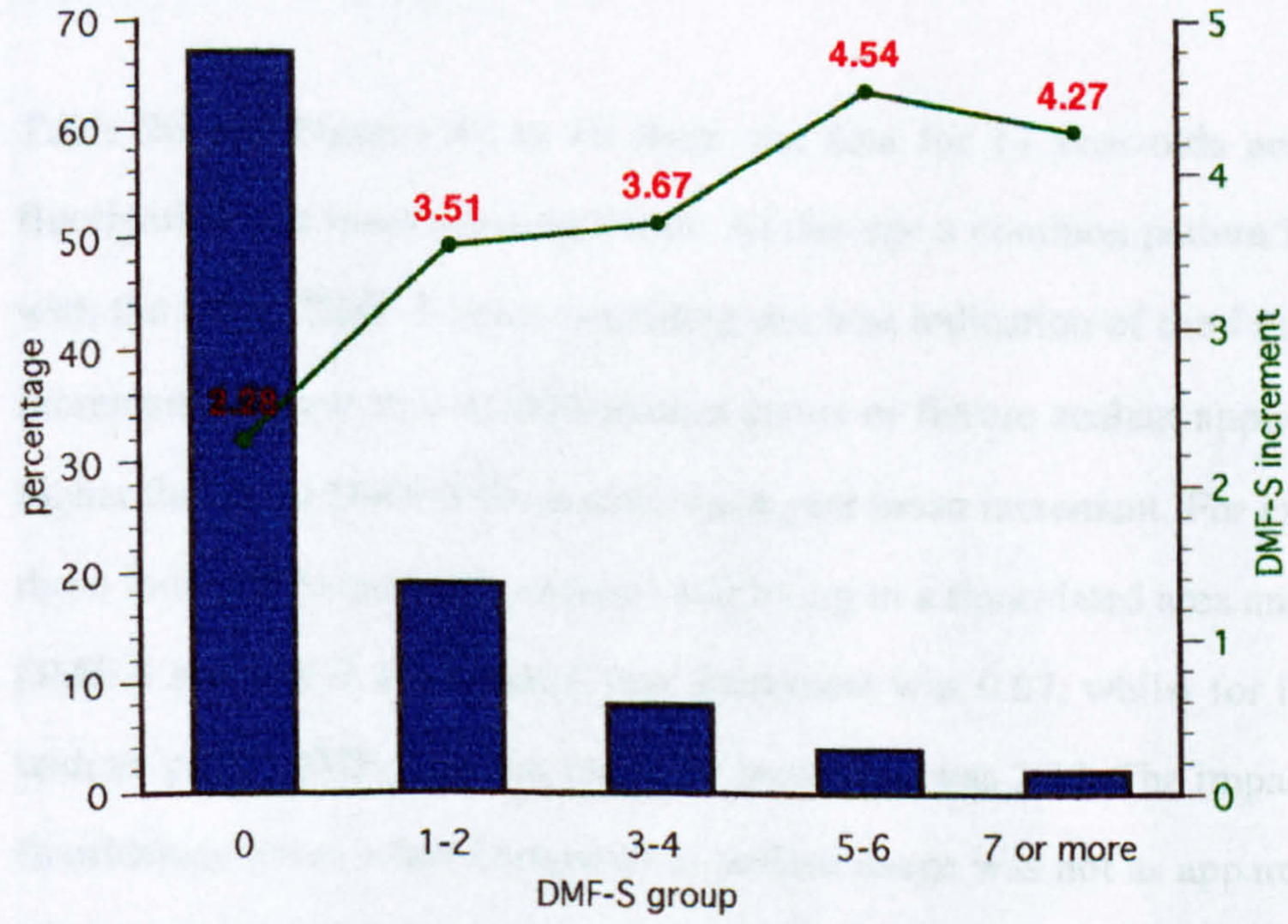


Figure 41: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 7 year-old children in non-fluoridated areas and not receiving fissure sealants



fluoridated areas the highest mean increment, 2.90, was found in the sub-group with an initial DMF-S score of between 1 and 2.

Table 19: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 7 year-old children according to fluoridation and sealant status.

DMF-S group	Fluoridated and sealants		Fluoridated, no sealants		Non-fluoridated and sealants		Non-fluoridated, no sealants	
	%	incrmt.	%	incrmt.	%	incrmt.	%	incrmt.
0	70	0.81	67	2.15	65	1.08	67	2.28
1-2	20	1.26	18	2.90	20	1.76	19	3.51
3-4	6	1.67	8	2.35	7	2.36	8	3.67
5-6	3	0.90	4	1.84	5	2.86	4	4.54
7 or more	1	1.33	2	1.04	3	2.64	2	4.27

Table 20 and Figures 42 to 45 show the data for 11 year-olds according to fluoridation and fissure sealant status. At this age a common pattern is apparent with the initial DMF-S score providing the best indication of the future disease increment. Irrespective of fluoridation status or fissure sealant application, the higher the initial DMF-S the greater the 4 year mean increment. For example, in those individuals receiving sealants and living in a fluoridated area and an initial DMF-S score of 0, the mean 4 year increment was 0.67, whilst for individuals with an initial DMF-S of 7 or more the increment was 2.38. The impact of water fluoridation status when compared to sealant usage was not as apparent as with the 7 year-olds. Whilst for those individuals with both preventive measures being used the mean increments were consistently lower when compared to those individuals with neither measure, the individuals receiving sealants but residing in

Figure 42: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 11 year-old children in fluoridated areas and receiving fissure sealants

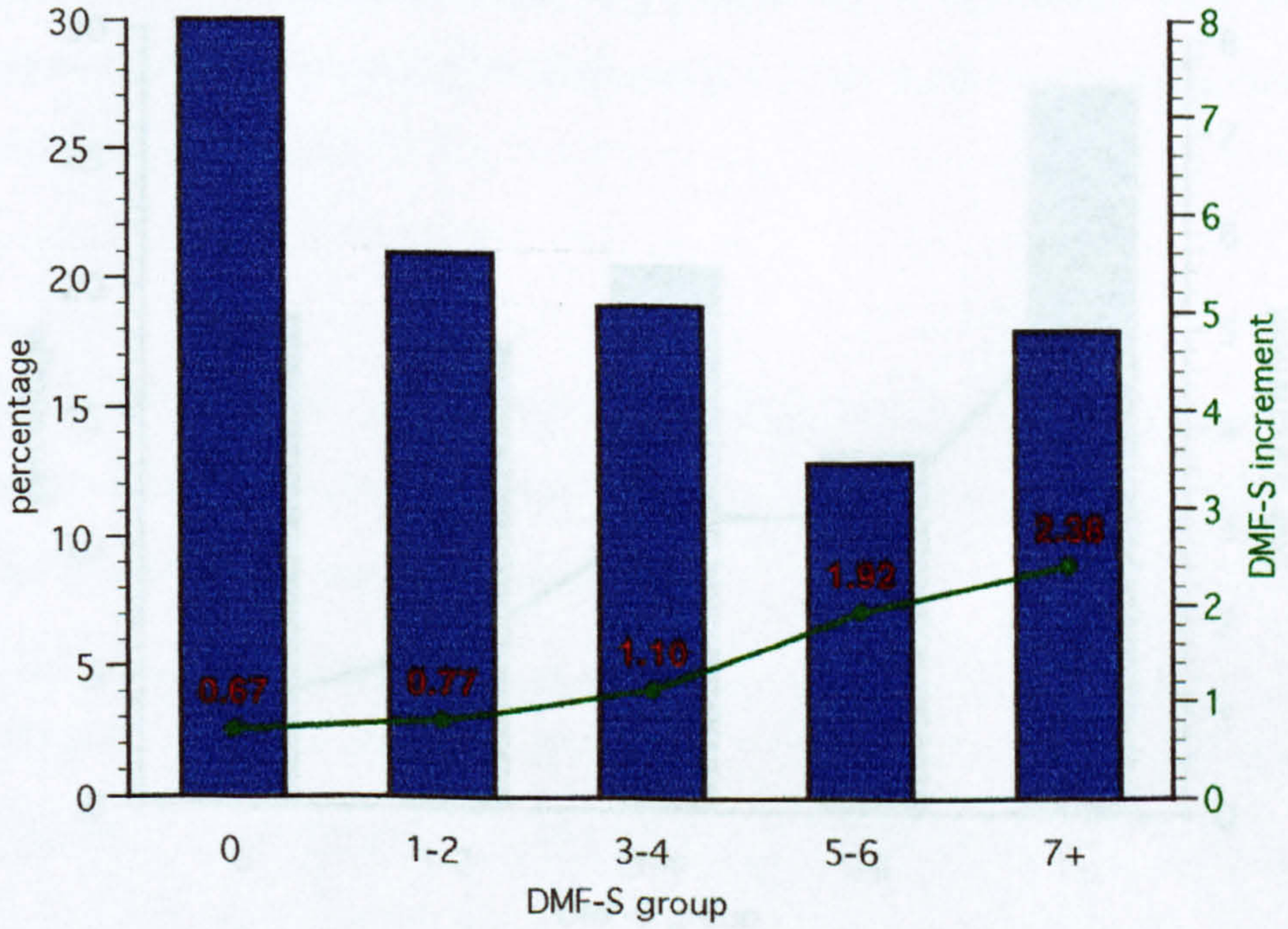


Figure 43: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 11 year-old children in fluoridated areas and not receiving fissure sealants

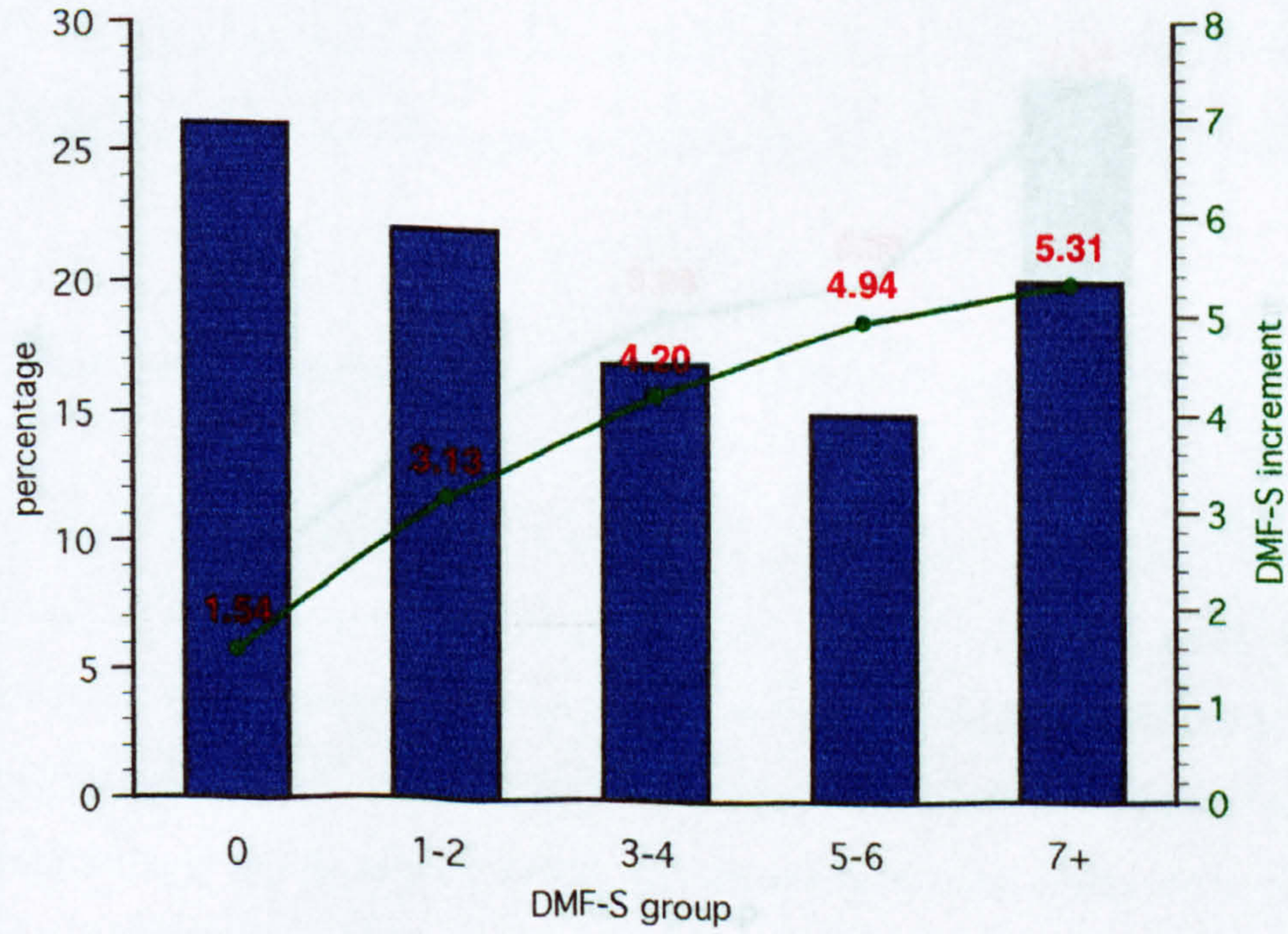


Figure 44: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 11 year-old children in non-fluoridated areas and receiving fissure sealants

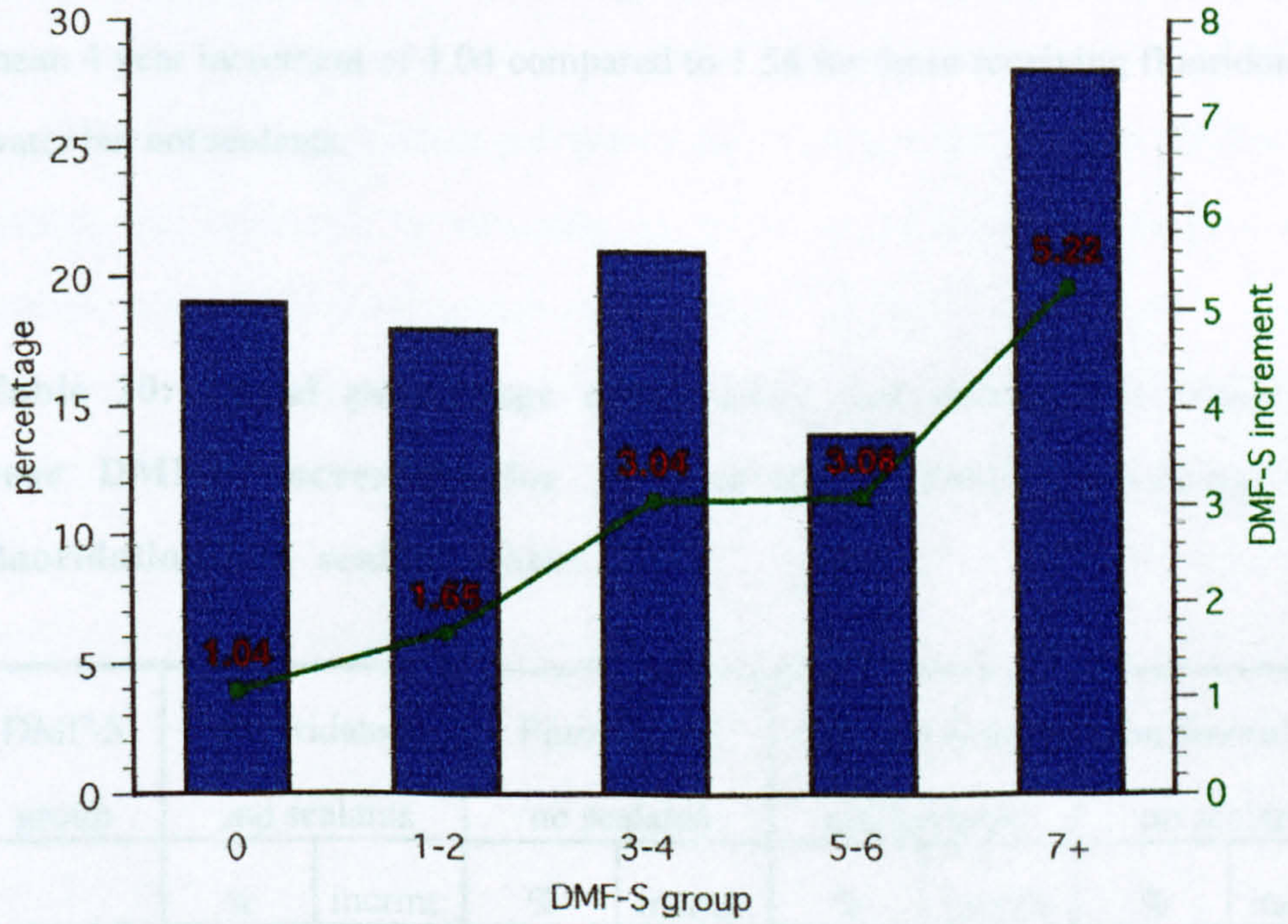
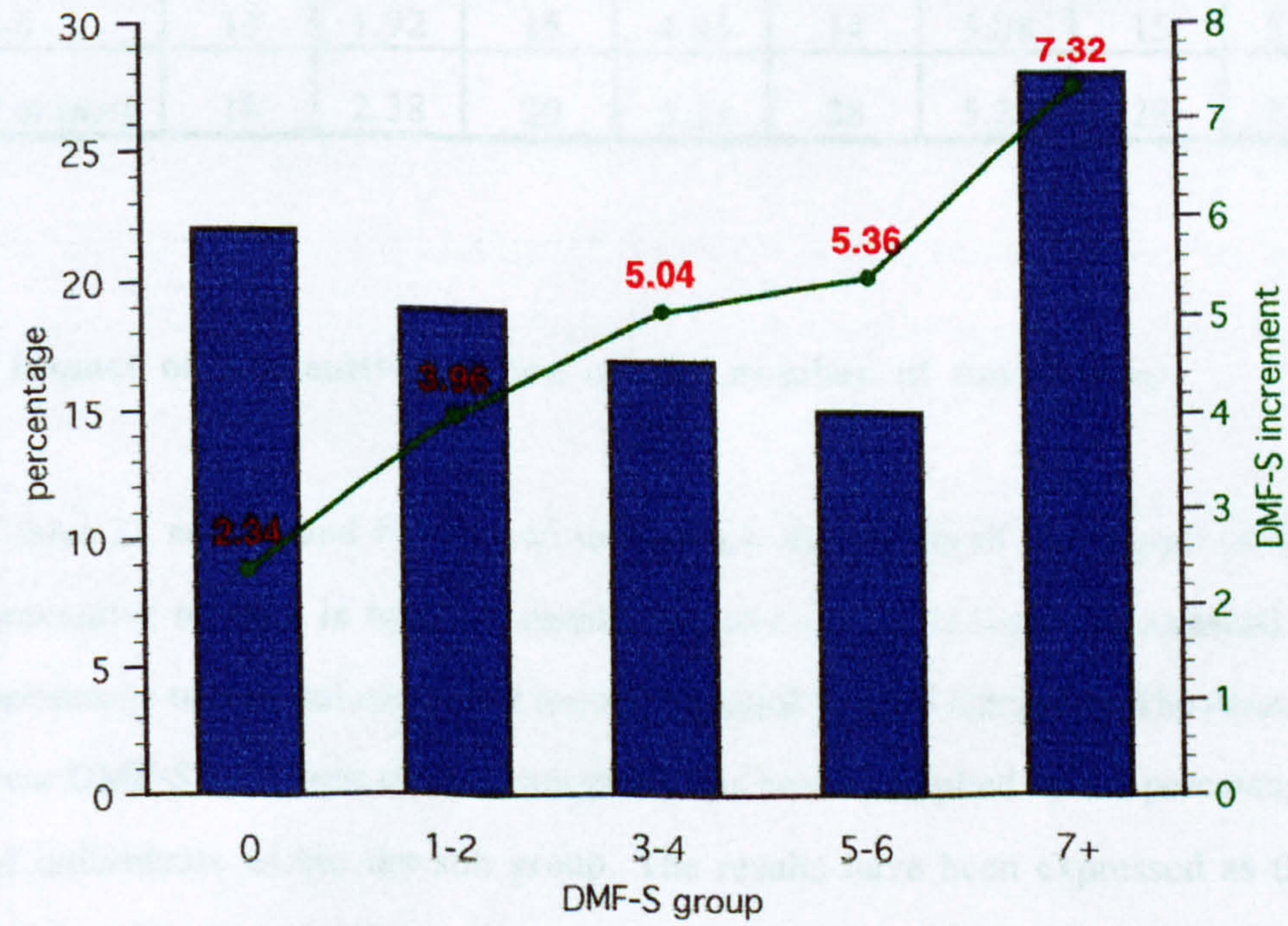


Figure 45: Initial percentage distribution and subsequent mean 4 year DMF-S increments for 11 year-old children in non-fluoridated areas and not receiving fissure sealants



a non-fluoridated area had lower mean increments when compared to those living in a fluoridated area but not receiving sealants. For example, for individuals with an initial DMF-S of 0, those receiving sealants but not fluoridated water had a mean 4 year increment of 1.04 compared to 1.54 for those receiving fluoridated water but not sealants.

Table 20: Initial percentage distribution and subsequent mean 4 year DMF-S increment for 11 year-old children according to fluoridation and sealant status.

DMF-S group	Fluoridated and sealants		Fluoridated, no sealants		Non-fluoridated and sealants		Non-fluoridated, no sealants	
	%	incrmt.	%	incrmt.	%	incrmt.	%	incrmt.
0	30	0.67	26	1.54	19	1.04	22	2.34
1-2	21	0.77	22	3.13	18	1.65	19	3.96
3-4	19	1.10	17	4.20	21	3.04	17	5.04
5-6	13	1.92	15	4.94	14	3.08	15	5.36
7 or more	18	2.38	20	5.31	28	5.22	28	7.32

4.6.2: Impact of preventive regime on the number of new lesions.

Tables 21 and 22 and Figures 46 to 53 show the results of the impact of the preventive regimes in terms of number of new carious lesions and cumulative percentage of the total number of lesions by initial DMF-S sub group. The mean 4 year DMF-S increment of each sub group has been multiplied by the percentage of individuals within the sub group. The results have been expressed as the number of lesions per 100 children. For example, the mean DMF-S increment for

the fluoridated sub group who received sealants and at an initial DMF-S of 0 was 0.81. The sub group at an initial DMF-S of 0 made up 70% of the total sub group of those individuals receiving fluoridated water and sealants, (Table 19). Thus the total number of lesions over a four year period within this sub group is 0.81 * 70, i.e. 57 lesions. For each of the preventive regime sub groups, the total number of lesions was calculated by summing the number of lesions from each of the various initial DMF-S sub groups. For example take the fluoridated individuals who received sealants, (Table 21). The number of lesions over the 4 year-period in the initial DMF-S sub group 0 would be 56.91, in the sub group 1-2, 25.18 lesions, in those at an initial DMF-S score of between 3-4, 10 lesions, 5-6, 2.69 lesions, and for those individuals with a DMF-S score of 7 or more, 1.33 lesions. The total number of lesions in this sub group is equal to 97. Thus, the percentage of lesions accounted for by the sub group at an initial DMF-S of 0 is 56.91 divided by 97, i.e. 59.21%.

Table 21 and Figures 46 to 49 show the number of new lesions and cumulative percentage for each of the preventive regimes according to initial DMF-S score for 7 year-olds. With the exception of those individuals in non-fluoridated areas and receiving sealants, over 50% of the total number of new lesions occurred in individuals with an initial DMF-S score of 0. Indeed, for all preventive regimes, the contribution made by individuals with the highest initial grouped DMF-S score, those with 7 or more lesions, was less than 6% of the total. For example, for those individuals receiving fluoridated water and sealants, the contribution to the total number of lesions made by those with an initial DMF-S of 7 or more was less than 2%.

Figure 46: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 7 year-old children in fluoridated areas and receiving fissure sealants over a 4 year period

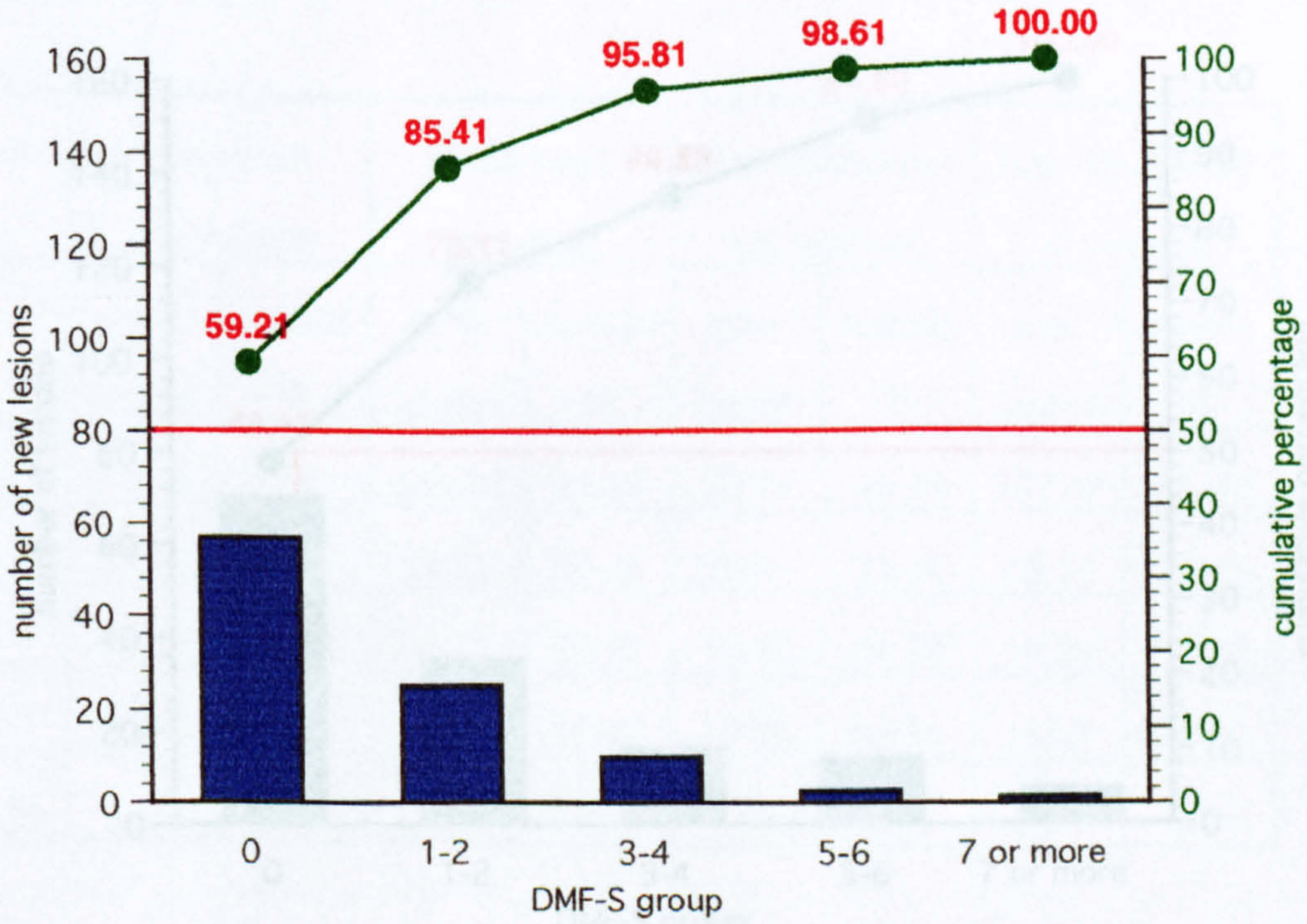


Figure 47: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 7 year-old children in fluoridated areas and not receiving fissure sealants over a 4 year period

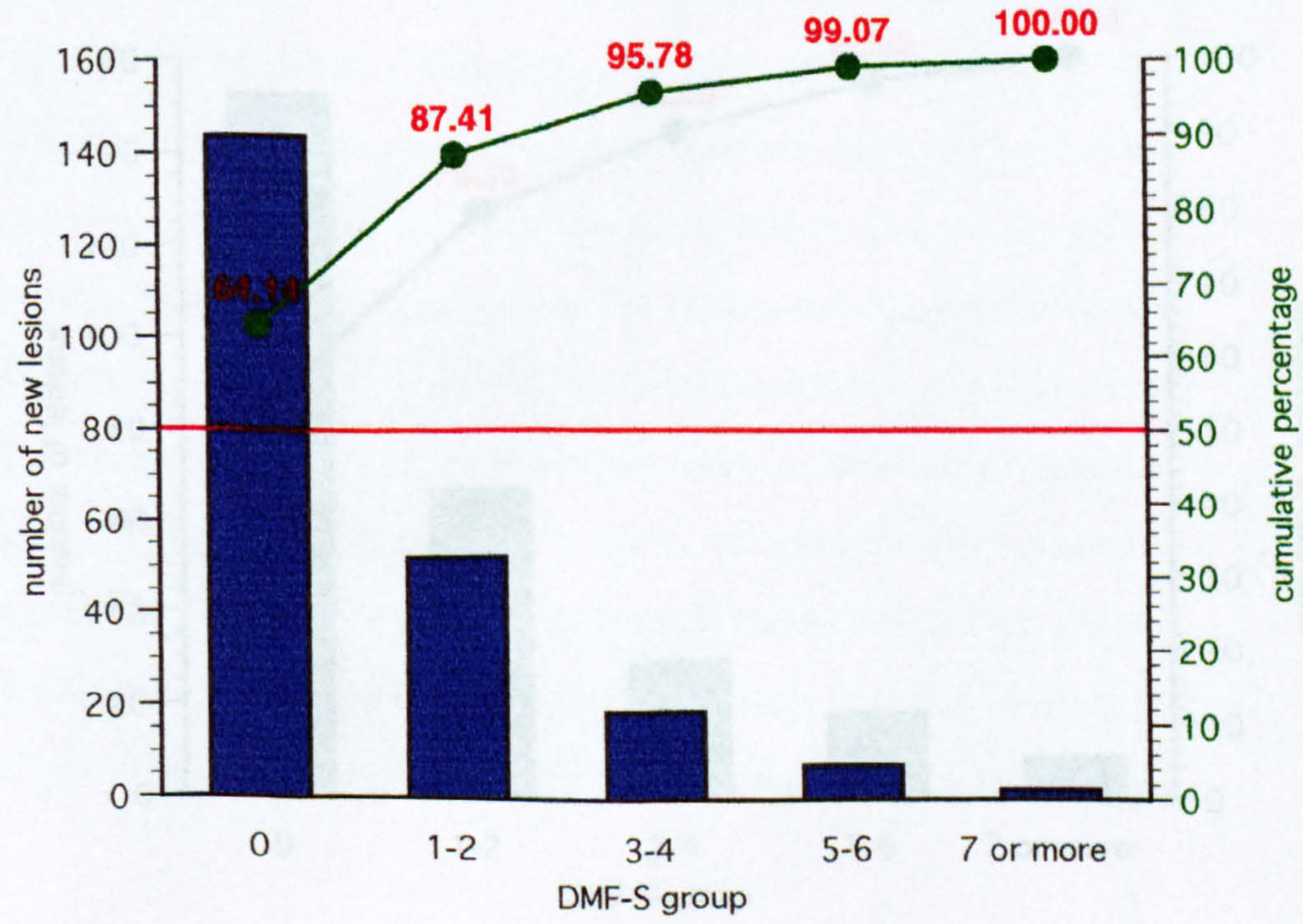


Figure 48: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 7 year-old children in non-fluoridated areas and receiving fissure sealants over a 4 year period

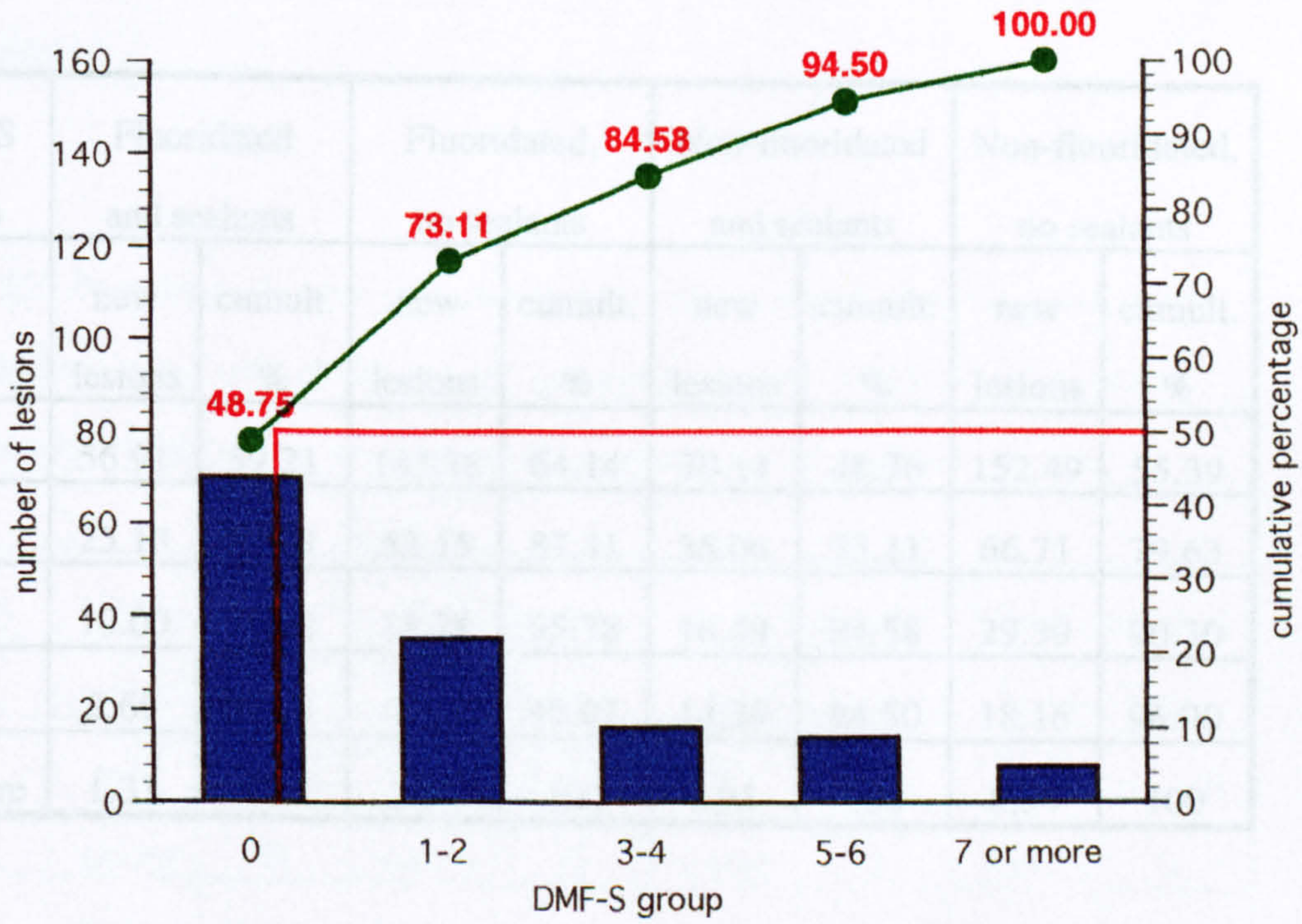


Figure 49: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 7 year-old children in non-fluoridated areas and not receiving fissure sealants over a 4 year period

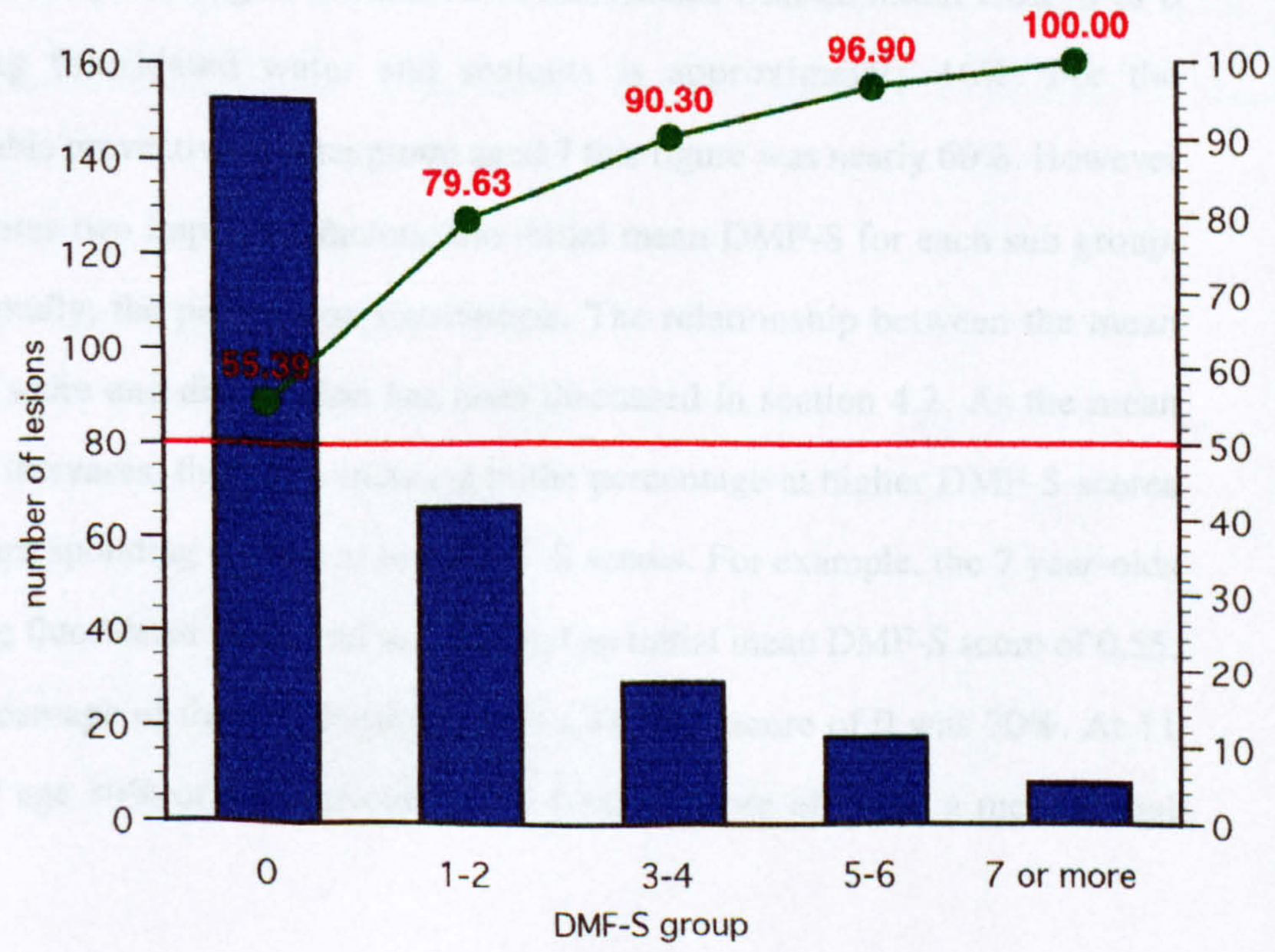


Table 21: Number of new carious lesions and cumulative percentage contribution per 100 children by grouped initial DMF-S score for 7 year-old children according to fluoridation and sealant status.

DMF-S group	Fluoridated and sealants		Fluoridated, no sealants		Non-fluoridated and sealants		Non-fluoridated, no sealants	
	new lesions	cumult. %	new lesions	cumult. %	new lesions	cumult. %	new lesions	cumult. %
0	56.91	59.21	143.78	64.14	70.14	48.76	152.49	55.39
1-2	25.18	85.41	52.15	87.41	35.06	73.11	66.71	79.63
3-4	10.00	95.81	18.78	95.78	16.49	84.58	29.39	90.30
5-6	2.69	98.61	7.38	99.07	14.29	94.50	18.16	96.90
7 or more	1.33	100	2.07	100	7.91	100	8.54	100

Table 22 and Figures 50 to 53 presents the data for 11 year-olds. At this age the contribution of the DMF-S groups at 0 is far smaller than at the earlier age. For example, the percentage contribution of individuals with an initial DMF-S of 0 receiving fluoridated water and sealants is approximately 16%. For the comparable preventive regime group aged 7 this figure was nearly 60%. However this ignores two important factors: the initial mean DMF-S for each sub group, and secondly, the percentage distribution. The relationship between the mean DMF-S score and distribution has been discussed in section 4.2. As the mean DMF-S increases, there is a increase in the percentage at higher DMF-S scores and a corresponding decline at low DMF-S scores. For example, the 7 year-olds receiving fluoridated water and sealants had an initial mean DMF-S score of 0.55. The percentage of those individuals with a DMF-S score of 0 was 70%. At 11 years of age 30% of 11 year-olds had a DMF-S score of 0 and a mean initial

DMF-S score of 2.09, whilst the corresponding percentage with an initial DMF-S score of 7 or greater was 1 and 18. The combined effects of the increased initial mean DMF-S and commensurate change in the percentage distribution, even for a similar mean increment, imply that the relative impact of each DMF-S sub group will change.

Table 22: Number of new carious lesions and cumulative percentage contribution per 100 children by grouped initial DMF-S score for 11 year-old children according to fluoridation and sealant status.

DMF-S group	Fluoridated and sealants		Fluoridated, no sealants		Non-fluoridated and sealants		Non-fluoridated, no sealants	
	new lesions	cumult. %	new lesions	cumult. %	new lesions	cumult. %	new lesions	cumult. %
0	19.95	15.98	40.01	11.10	19.72	6.52	51.50	10.35
1-2	16.23	28.98	68.84	30.20	29.70	16.34	75.22	25.46
3-4	20.94	45.75	71.47	50.02	63.90	37.46	85.70	42.67
5-6	24.95	65.73	74.06	70.57	43.05	51.69	80.39	58.82
7 or more	42.79	100.00	106.10	100.00	146.16	100.00	205.02	100.00

If the approach adopted was to identify those individuals with the higher DMF-S scores, i.e. 7 or more, at the commencement of the programme, the policy would be ignoring groups in which more than 50% of the total number of lesions would occur, (Table 21 and 22).

Figure 50: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 11 year-old children in fluoridated areas and receiving fissure sealants over a 4 year period

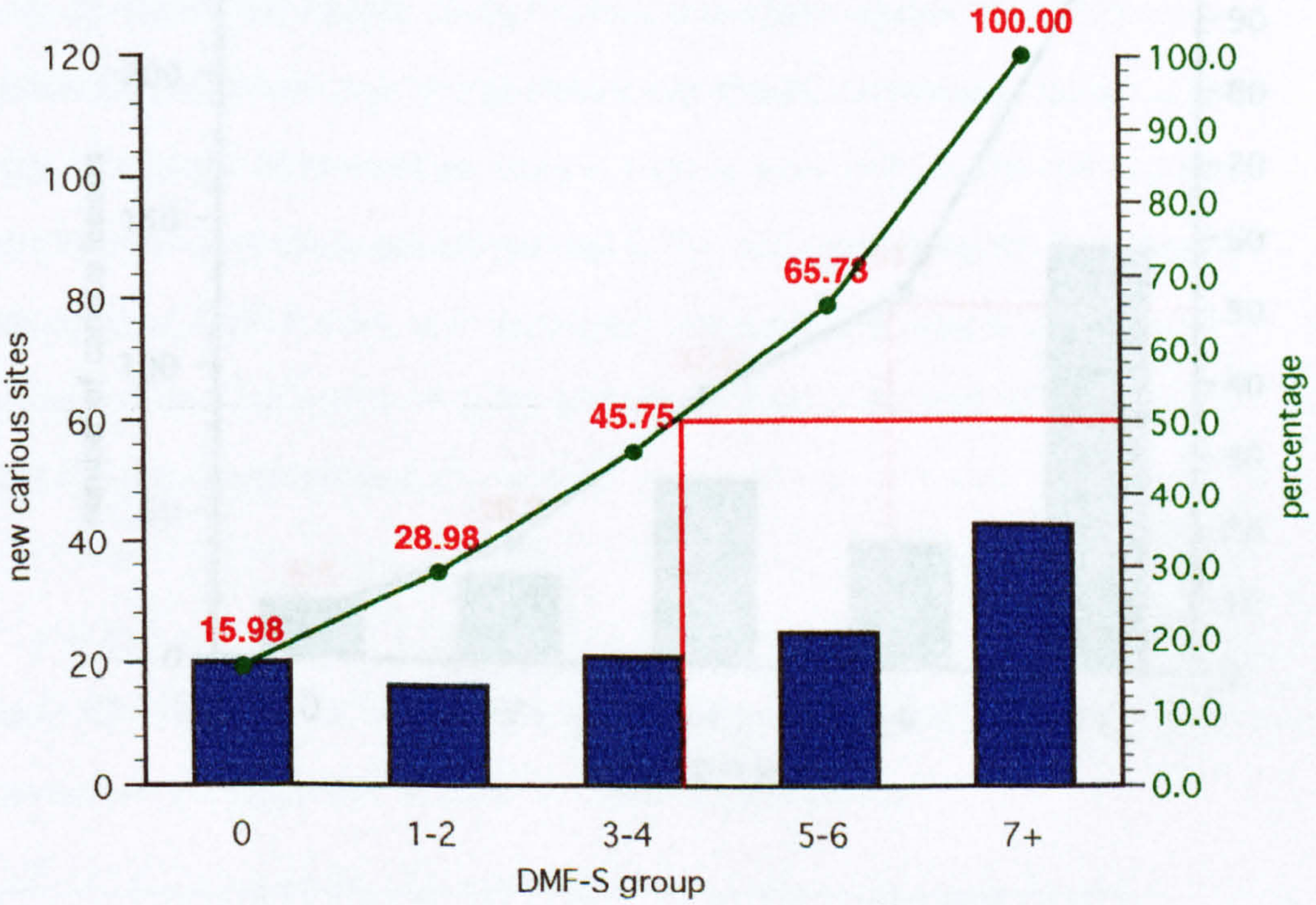


Figure 51: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 11 year-old children in fluoridated areas and not receiving fissure sealants over a 4 year period

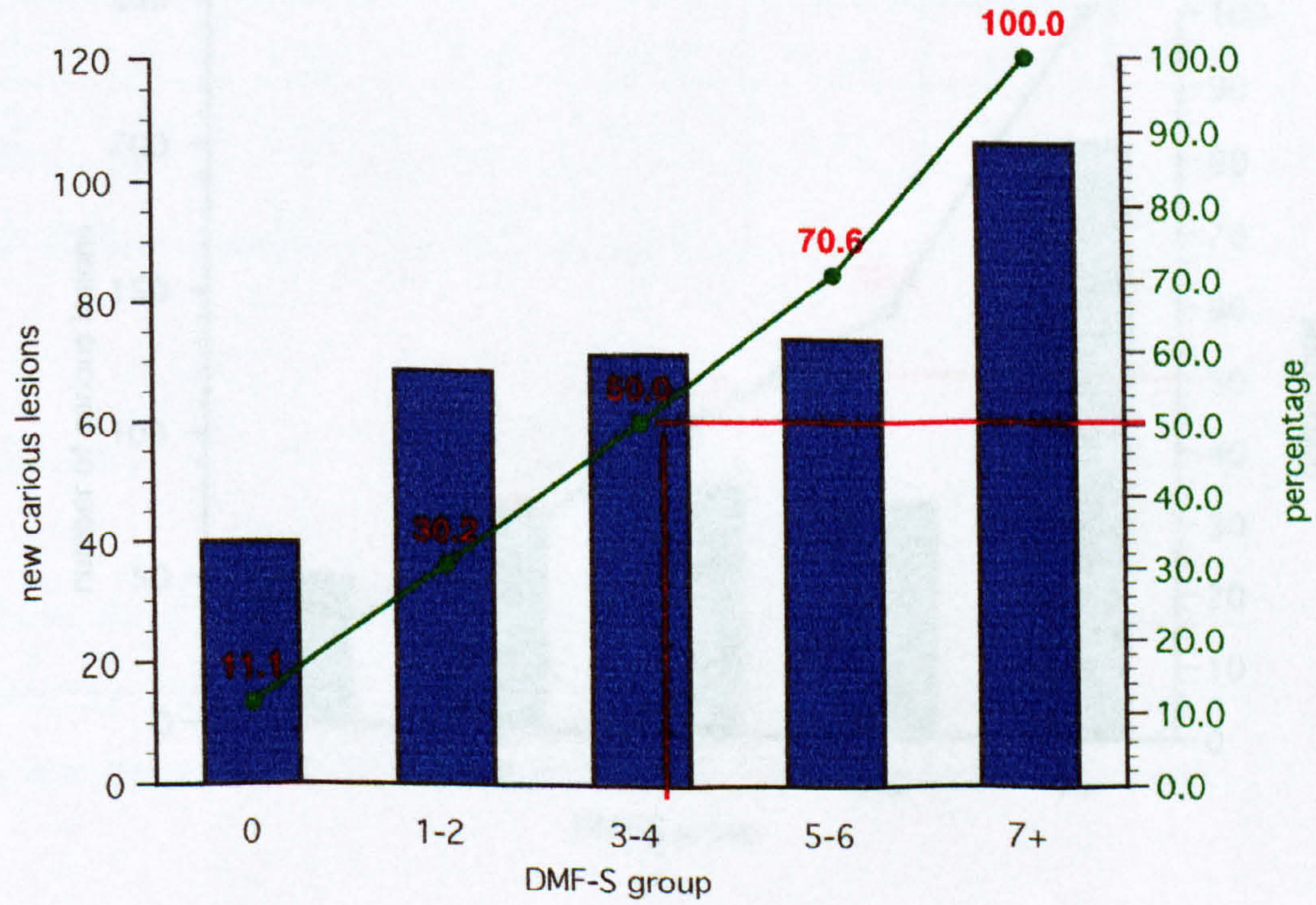


Figure 52: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 11 year-old children in non-fluoridated areas and receiving fissure sealants over a 4 year period

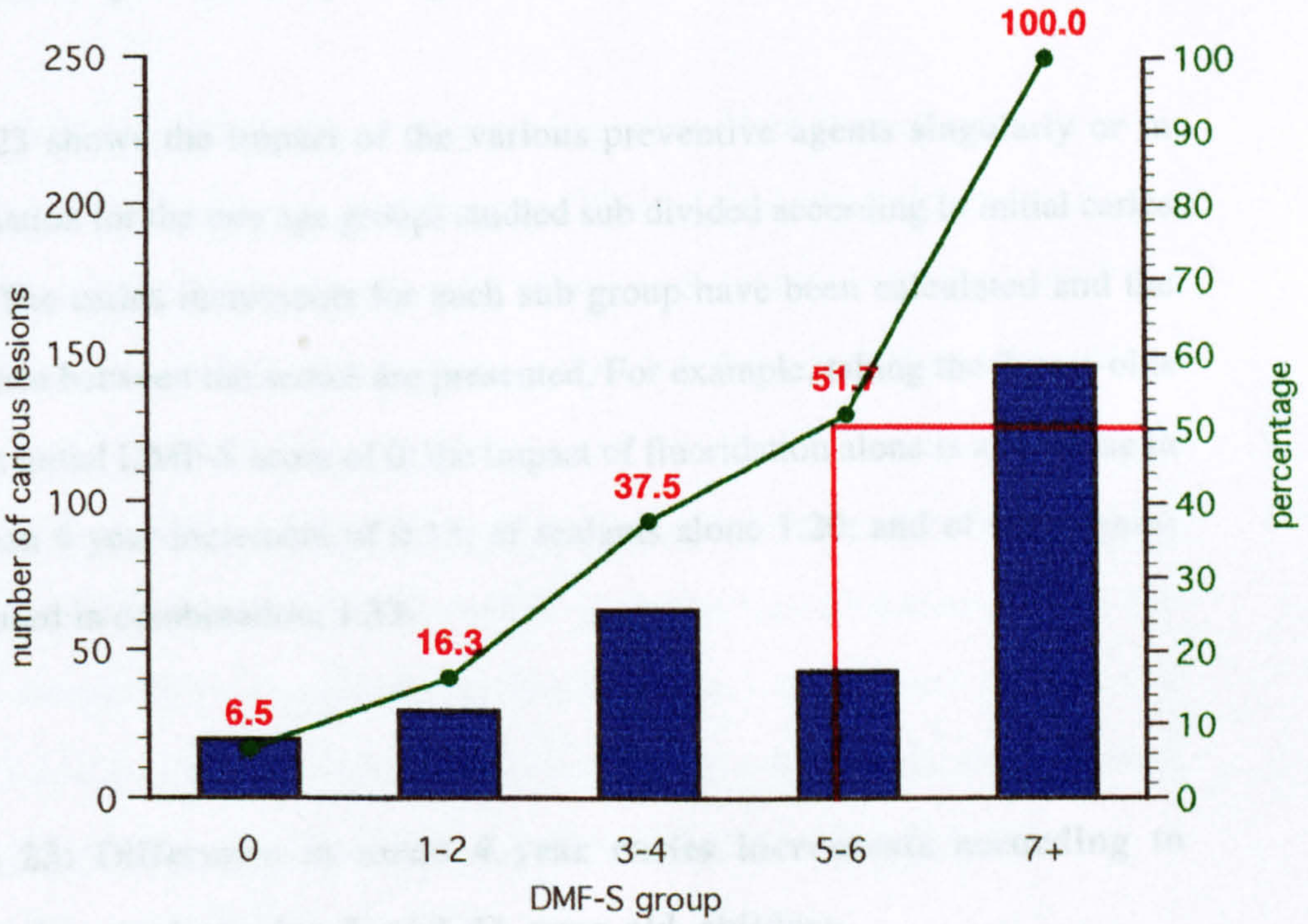
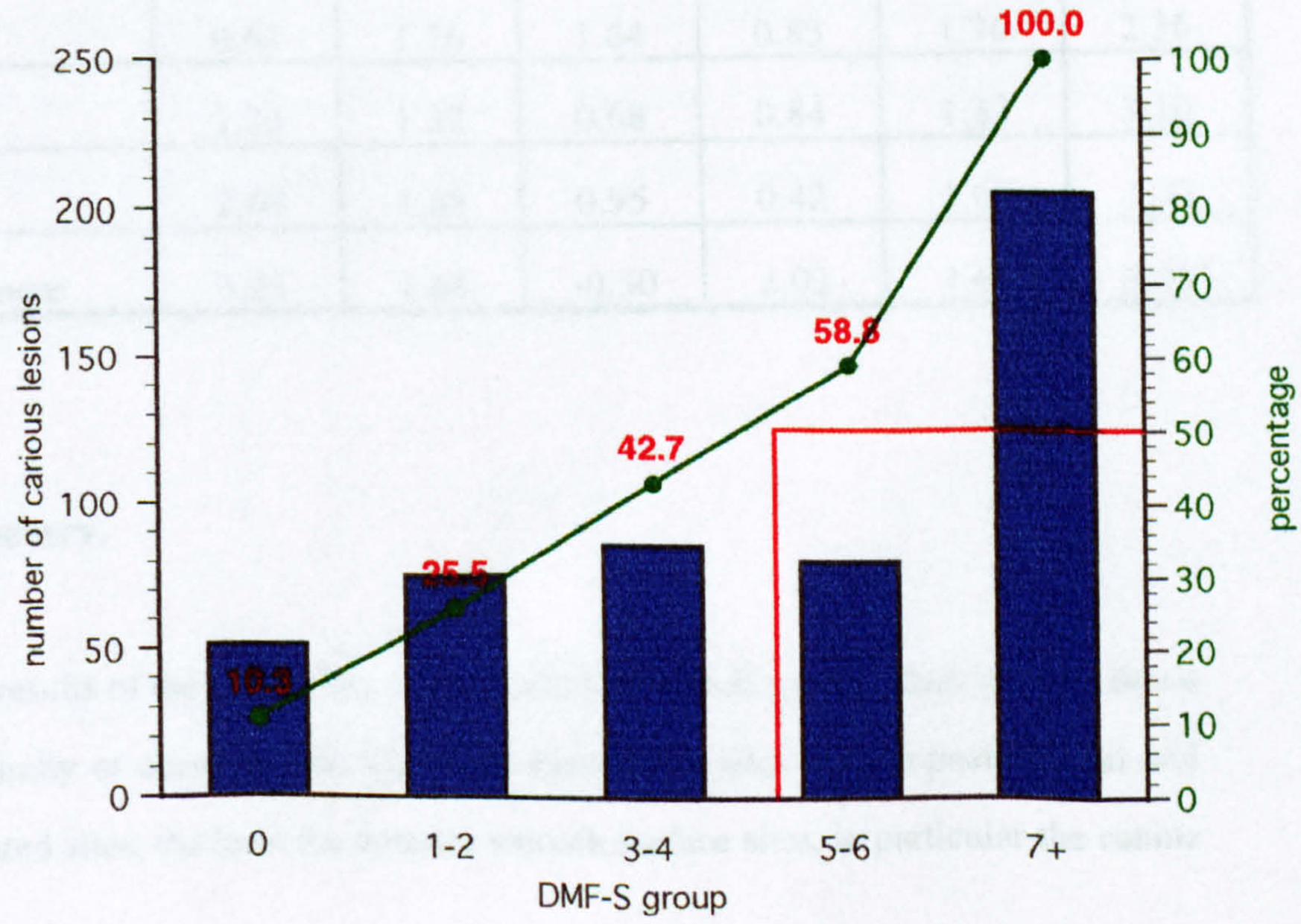


Figure 53: Number of new carious lesions per 100 children by grouped DMF-S score and cumulative percentage for 11 year-old children in non-fluoridated areas and not receiving fissure sealants over a 4 year period



4.6.3: Impact of preventive package on caries increments.

Table 23 shows the impact of the various preventive agents singularly or in combination for the two age groups studied sub divided according to initial caries score. The caries increments for each sub group have been calculated and the difference between the scores are presented. For example, taking the 7 year-olds with an initial DMF-S score of 0: the impact of fluoridation alone is a decrease in the mean 4 year-increment of 0.13; of sealants alone 1.20; and of both agents when used in combination, 1.33.

Table 23: Difference in mean 4 year caries increments according to preventive package for 7 and 11 year-old children.

	7 year-olds			11 year-olds		
DMF-S group	F-	sealants	sealant(F)	F-	sealants	sealant(F)
0	0.13	1.20	1.33	0.80	1.20	0.87
1-2	0.61	1.76	1.64	0.83	1.76	2.36
3-4	1.33	1.32	0.68	0.84	1.32	3.10
5-6	2.69	1.68	0.95	0.42	1.68	3.02
7 or more	3.23	1.64	-0.30	2.02	1.64	2.93

4.7: Summary.

The results of the probability of individual sites undergoing attack indicate that a hierarchy of attack exists. The most susceptible sites are the posterior pit and fissured sites, the least the anterior smooth surface sites, in particular the canine

tooth surfaces. In addition there is a symmetry about the susceptibilities, both in a vertical plane, and, for the posterior sextants, in a horizontal plane.

The distribution of probabilities is also independent of the demographic variables studied; sex, ethnicity, age and fluoride. This suggests that, for sex and age, differences in the levels of caries may be due to the number of sites erupted but that the order in which the sites become carious is independent of these variables.

The other important relationship is that between fluoride and site susceptibility. The impact of fluoride is also independent of site types; all sites benefit. The order of site susceptibility has not changed, which one would expect to happen if fluoride had a more beneficial impact of some sites when compared to others. Those with the least propensity for caries benefit first.

When combined with the results in section 4.2, these results indicate that the development of an accurate caries progression model is possible. In section 4.2 the distribution was normal in shape, and a mathematical relationship exists between the mean and variance. The results in this section indicate that the order of site susceptibility can be modelled. Thus, if the distribution within a population can be ascertained, the number of individuals at a particular DMF-S and the distribution of caries within each individual can be modelled.

5. VERIFICATION OF THE CARIES CONCEPTUAL MODEL

5.1: Introduction

The conceptual model in Section 4 identified a number of key relationships. The data used were those from the NPDDP study collected in the late seventies in the United States. The question that arises is whether the relationships identified still hold for current caries data. This section will test the findings of the analyses in Section 4 with data from studies carried out by the British Association for the Study of Community Dentistry (BASCD) and a 4 year longitudinal study carried out by Walsall Health Authority in conjunction with the Welsh School of Medicine and Dentistry. Each of the four aspects identified from the NPDDP will be tested. These are:

i. the relationship between caries prevalence and mean DMF score,

ii. the relationship between mean caries score and the associated variance,

iii. the relationship between the mean DMF score and the distribution at each DMF score, and

iv. the hierarchical nature of the caries attack.

5.2: The relationship between caries prevalence and mean DMF score

The NPDDP data inferred that a relationship existed between the prevalence of dental caries and the mean DMF score. For the NPDDP data, the relationship between DMF-T and prevalence of caries could be expressed by the formula $y = 42.0 * x^{0.51}$.

The formula describing caries prevalence and mean DMF-T collected for the BASCD 12 year-olds was $y = 41.8 * x^{0.64}$, and for the 14 year-olds $y = 41.6 * x^{0.52}$, (Figures 54 and 55). These two figures are remarkably similar despite the differing examination conditions, including examiners and criteria, and the dates when the data were collected. This suggests that the mathematical relationship between caries prevalence and mean DMF-T is fixed: for any given DMF-T the percentage of individuals having caries within the population will be known and vice versa.

5.3: The relationship between mean caries score and the associated variance

The relationship between the mean caries score and the associated standard deviation for the BASCD 12 and 14 year-old data are shown in Figures 56 and 57. Despite the age difference between the two data sets there is again a remarkable similarity in the two mathematical formulae describing the relationship between the mean caries score and its associated standard deviation. For the 12 year-olds the formula is $y = 1.53 * x^{0.55}$, where y = standard deviation, and x = mean DMF-T score, and for the 14 year-olds, $y = 1.63 * x^{0.58}$.

These formulae compare to $y = 1.63 * x^{0.71}$ for the NPDDP 12 year-olds, and $y = 1.60 * x^{0.72}$ for the 14 year-olds. The small degree of variation between the BASCD and NPDDP could be explained by the differing criteria used: with the more sensitive criteria in the NPDDP smaller lesions are more likely to be classified as positive. This would not affect the overall relationship between prevalence and DMF scores, as both the prevalence and mean scores will be affected by any difference in a dichotomous decision. However, for the mean and

Figure 54: The relationship between percentage of sample with decay experience and mean DMF-T for 12 year-old children

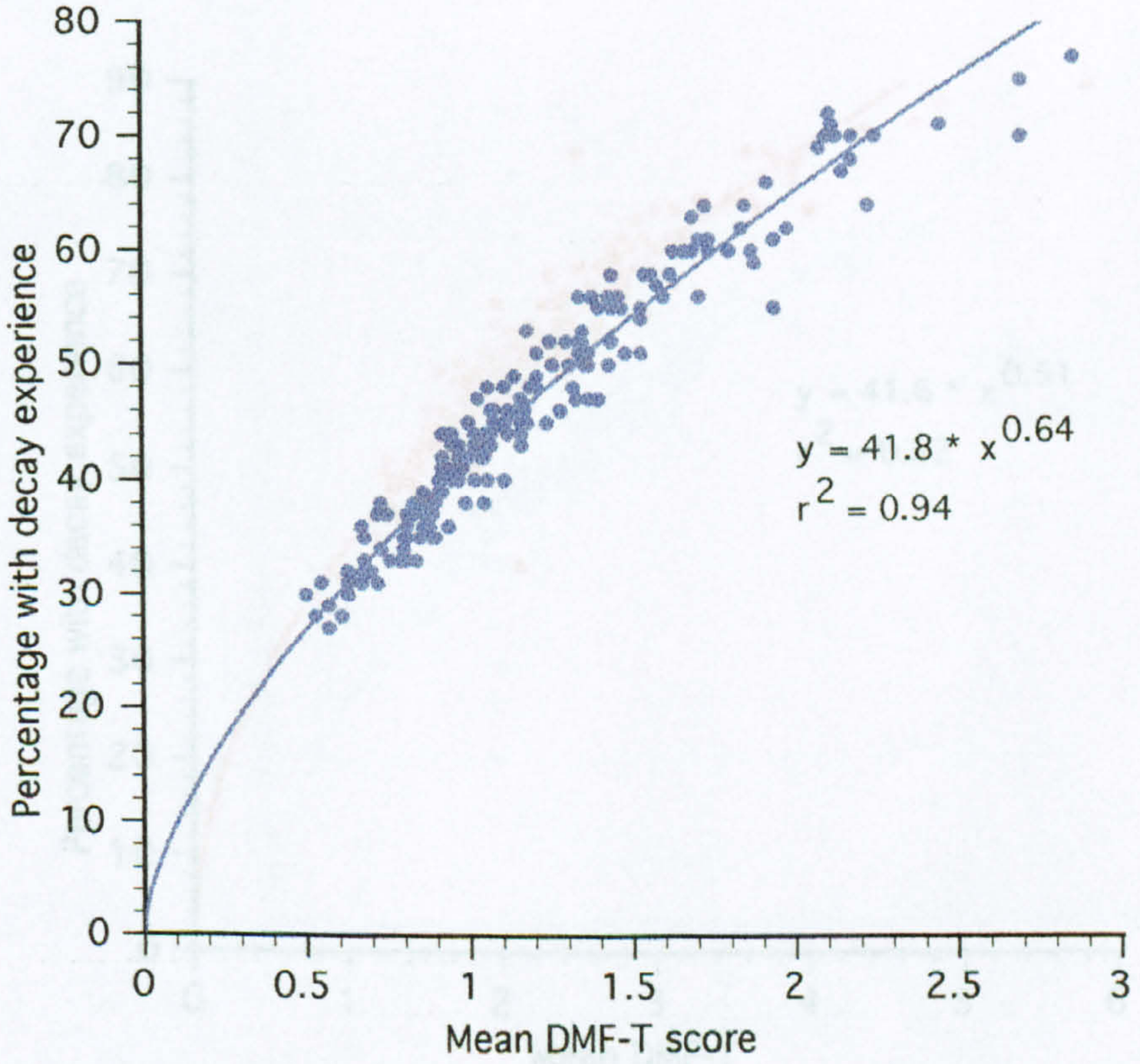


Figure 55: The relationship between percentage of sample with decay experience and mean DMF-T for 14 year-olds

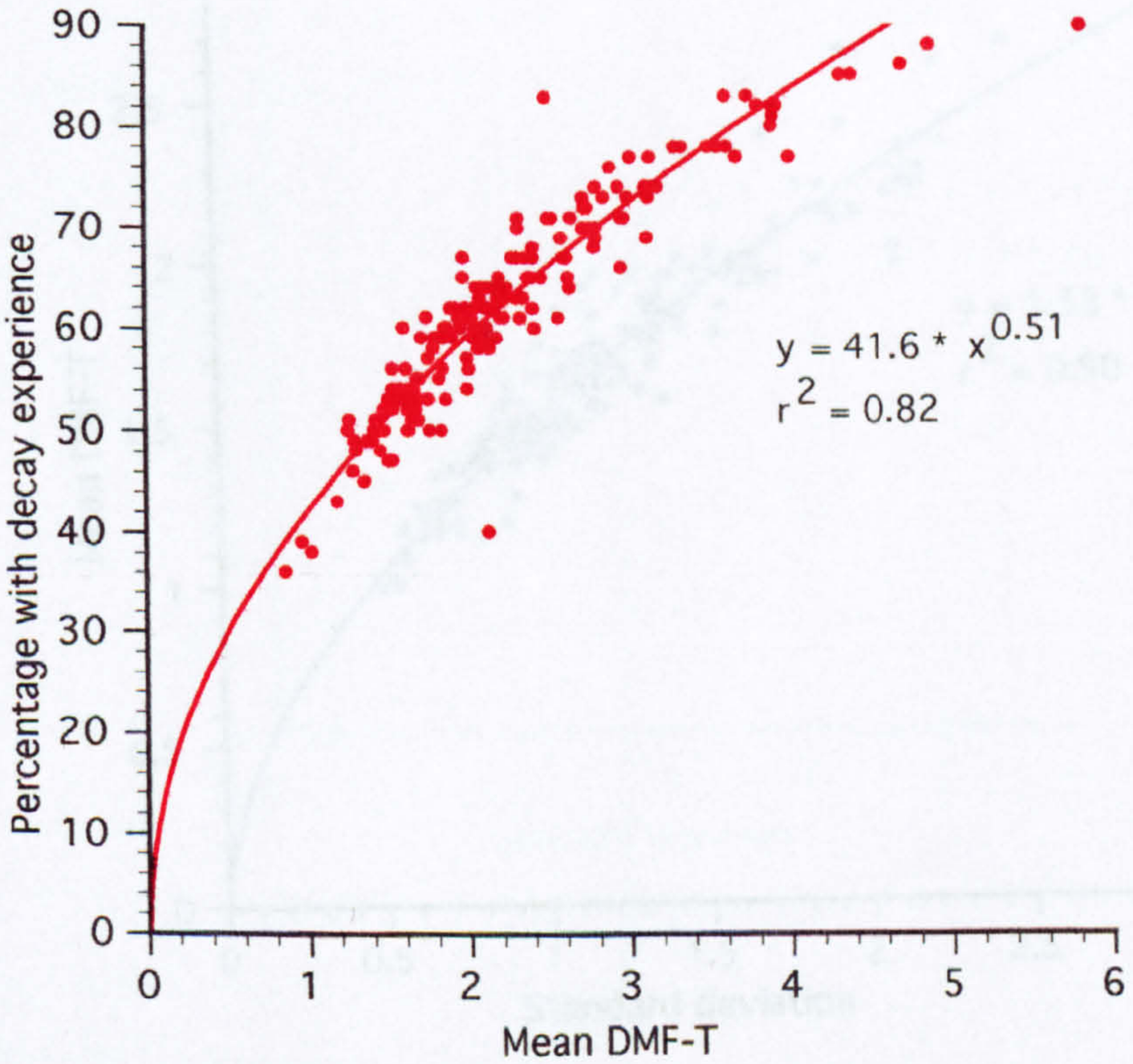


Figure 56: The relationship between the mean DMF-T score and the standard deviation for 12 year-old children

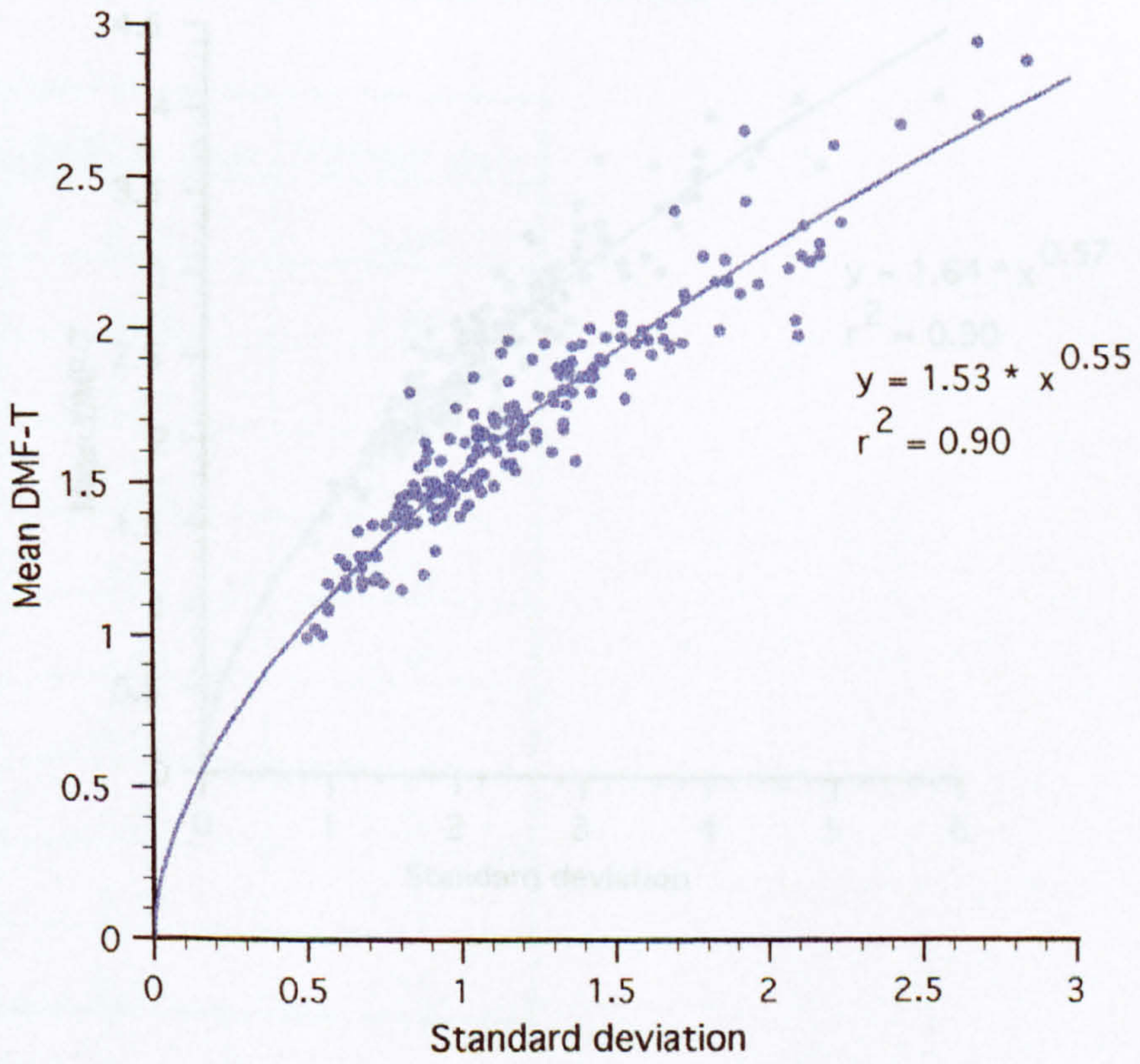
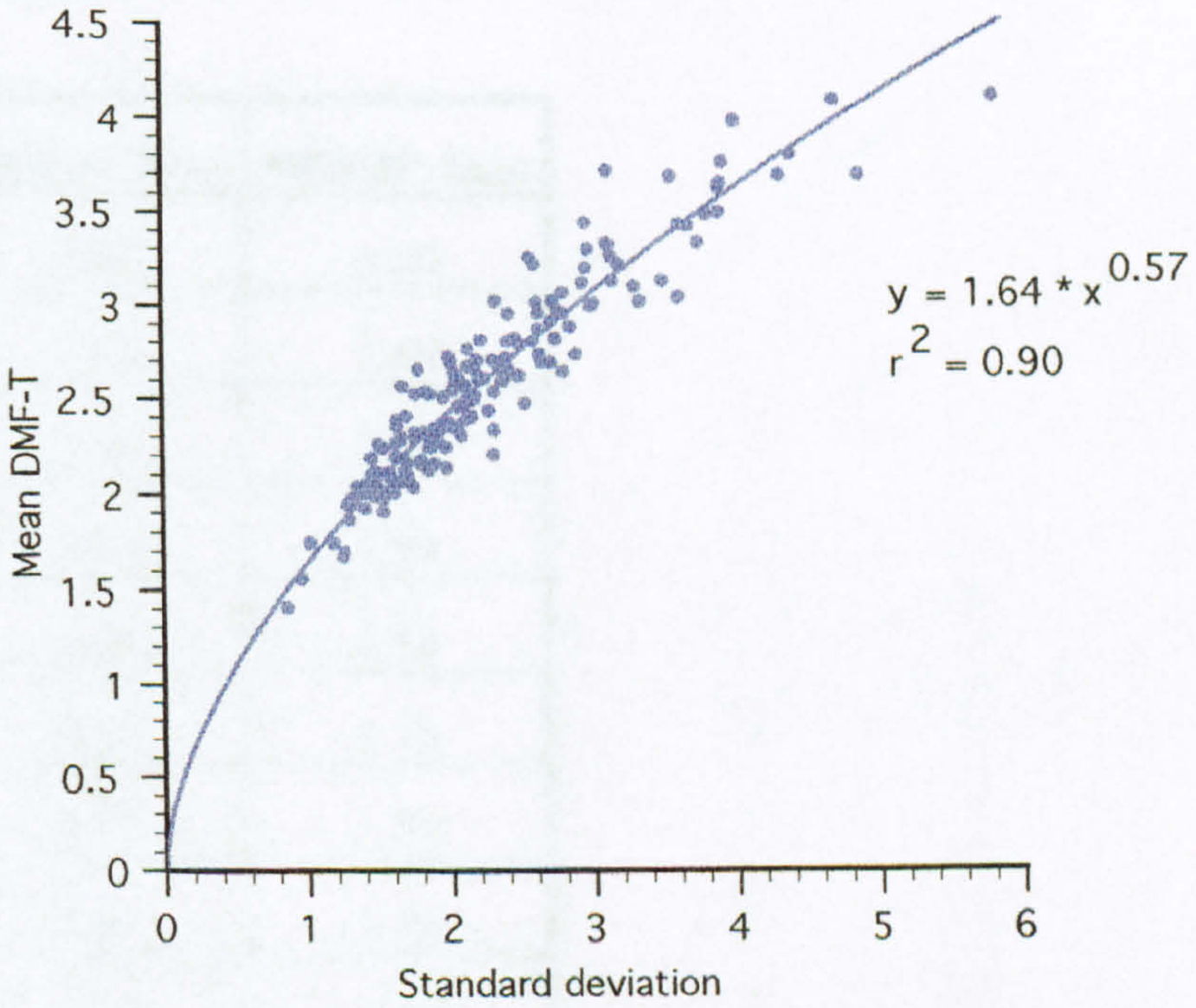


Figure 57: The relationship between the mean DMF-T score and the standard deviation for 14 year-olds



variance, the higher number of lesions are likely to give a wider variance, and in consequence, a larger standard deviation for a given mean.

Table 24: The difference between the formula used for reporting the relationship between the mean DMF-T and standard deviation for using data from BASCD 14 year-olds and NPDDP 14 year-olds.

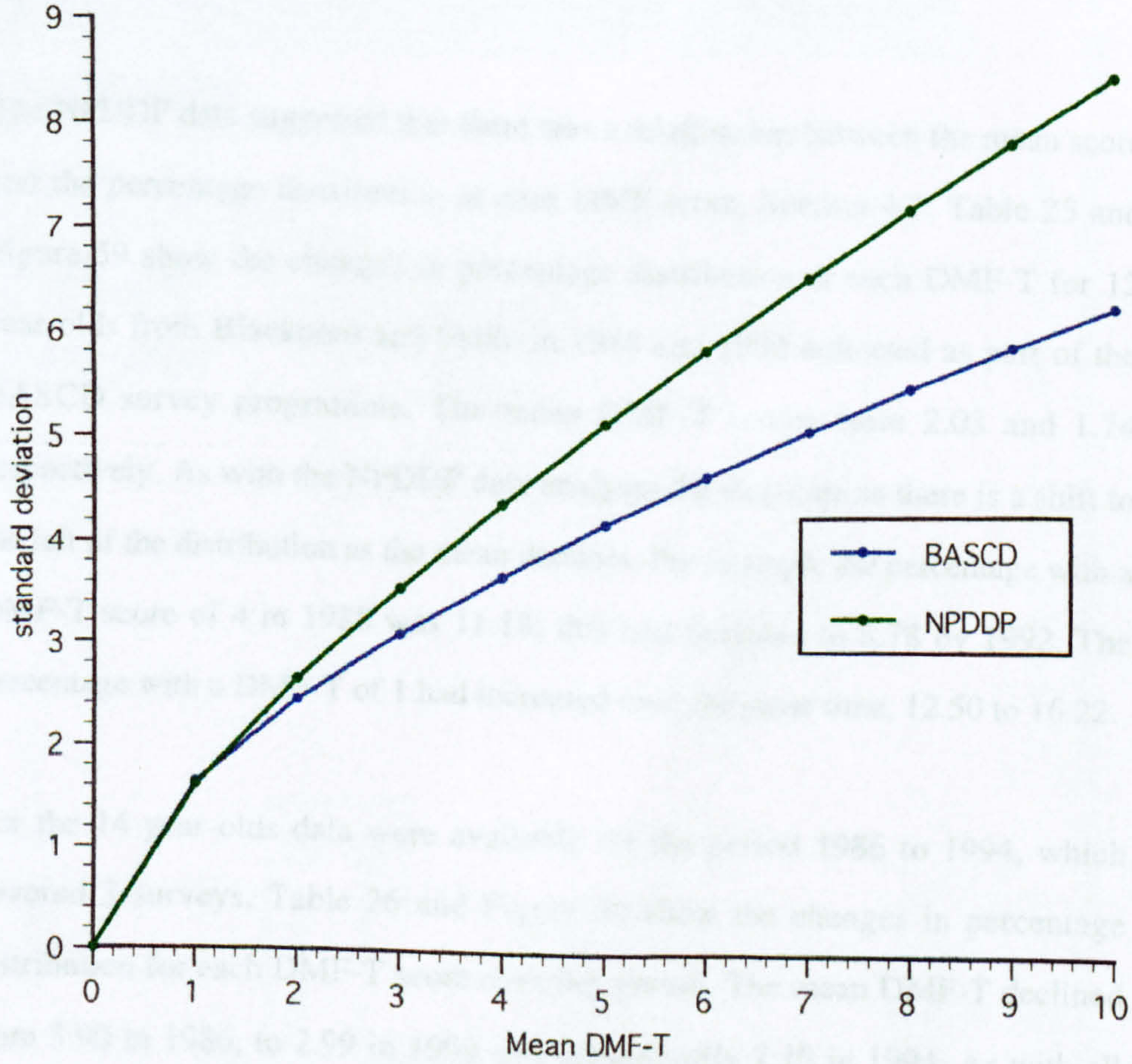
DMF-T	BASCD data	NPDDP data
0	0.00	0.00
1	1.63	1.60
2	2.44	2.64
3	3.08	3.53
4	3.64	4.34
5	4.15	5.10
6	4.61	5.81
7	5.04	6.50
8	5.44	7.15
9	5.83	7.78

Differences not significant ($p > 0.05$)

Despite theses differences in the formulae, the actual magnitude in terms of teeth units is small, (Table 24 and Figure 58). For example for the 14 year-olds, the BASCD formula would suggest at a mean DMF-T of 3, a relatively high level of caries in the UK today, the difference in standard deviation 'teeth units' is only 0.45.

This suggests that, whilst variation in the relationship between the mean and standard deviation may occur with differing examination criteria, that a

Figure 58: The conceptual model relationship between mean DMF-T and standard deviation for using data from BASCD and NPDDP 14 year-old children



mathematical formula can be used to describe the relationship. For the same criteria, the formula are independent of age.

5.4: The relationship between the mean DMF score and the distribution at each DMF score.

The NPDDP data suggested that there was a relationship between the mean score and the percentage distribution at each DMF score, Section 4.7. Table 25 and Figure 59 show the changes in percentage distribution at each DMF-T for 12 year-olds from Blackpool and Fylde in 1988 and 1992 collected as part of the BASCD survey programme. The mean DMF-T scores were 2.03 and 1.74 respectively. As with the NPDDP data analyses the distribution there is a shift to the left of the distribution as the mean declines. For example the percentage with a DMF-T score of 4 in 1988 was 11.18; this had declined to 8.78 by 1992. The percentage with a DMF-T of 1 had increased over the same time, 12.50 to 16.22.

For the 14 year-olds data were available for the period 1986 to 1994, which covered 3 surveys. Table 26 and Figure 60 show the changes in percentage distribution for each DMF-T score over this period. The mean DMF-T declined from 3.90 in 1986, to 2.99 in 1990 and subsequently 2.10 in 1994. As with all the distributions reported to date, there was a shift to the left of the population. For example, the percentage found at a DMF-T of 9 in 1986 was 4.02. This declined to 2.39% in 1990 and then to 1.71% in 1994. Conversely the percentages caries free increased over this period: 14.86 in 1986, 27.14 in 1990 and 34.81 in 1994.

Tables 27 and 28 and Figures 61 and 62 show the 2 year longitudinal changes in the 12 year-old populations in 1988 and 1992. Both distributions show a shift to

Figure 59: **Changes in the percentage distribution of DMF-T scores for individuals aged 12 from 1988 to 1992.**

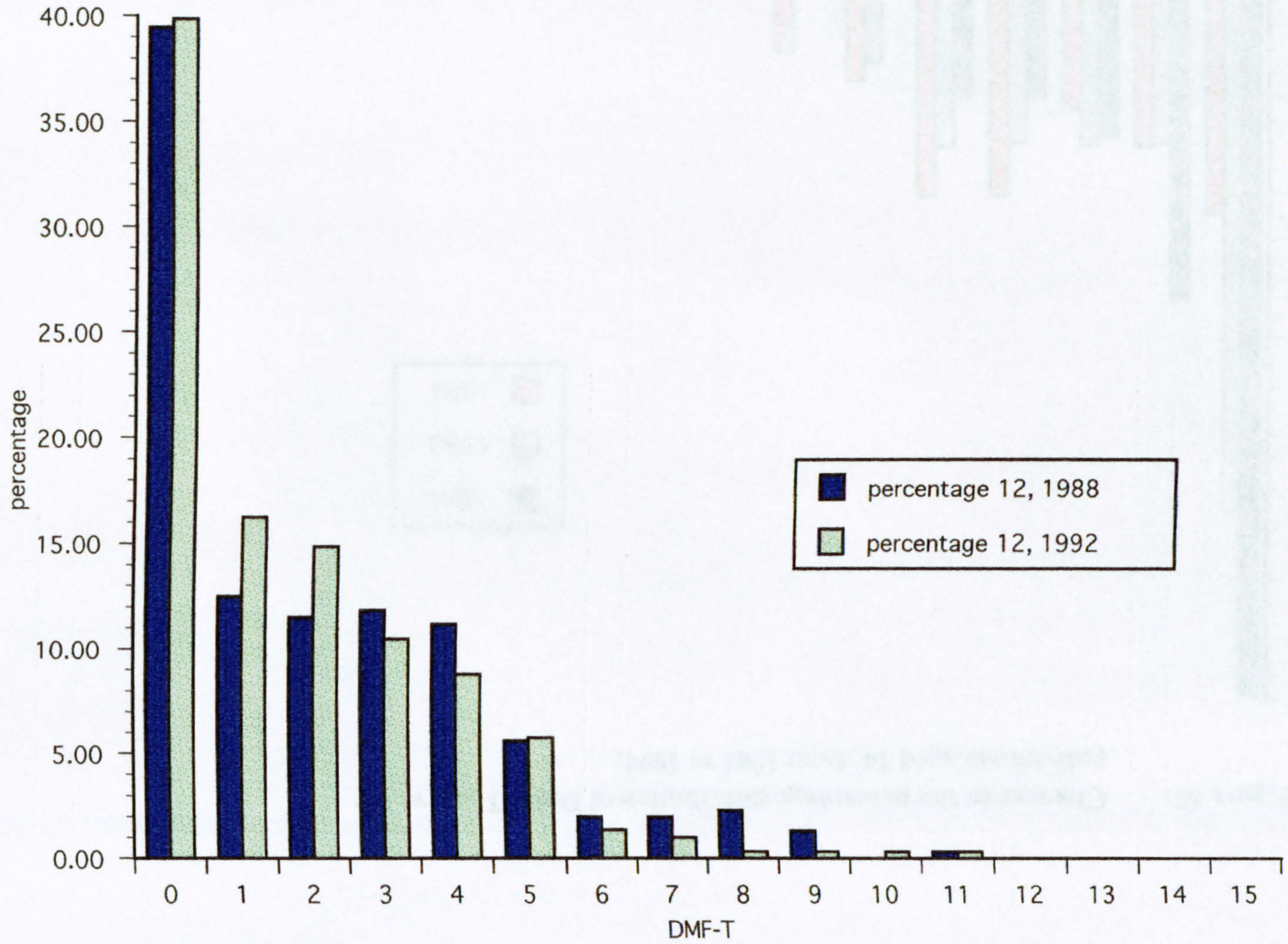


Figure 60: **Changes in the percentage distribution of DMF-T scores for individuals aged 14 from 1986 to 1994.**

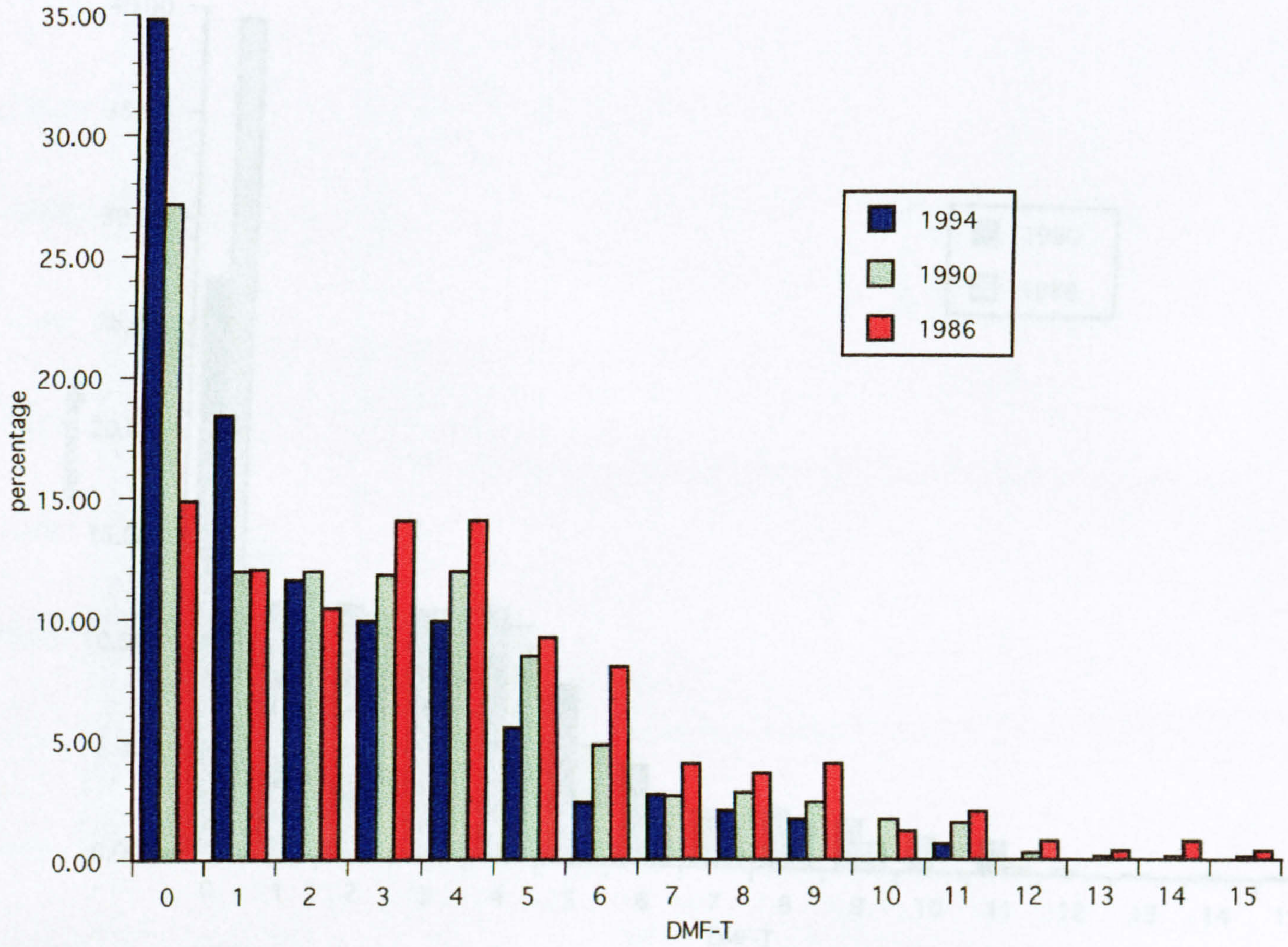


Figure 61: **Two year change in the percentage distribution of DMF-T scores for individuals aged 12 in 1988.**

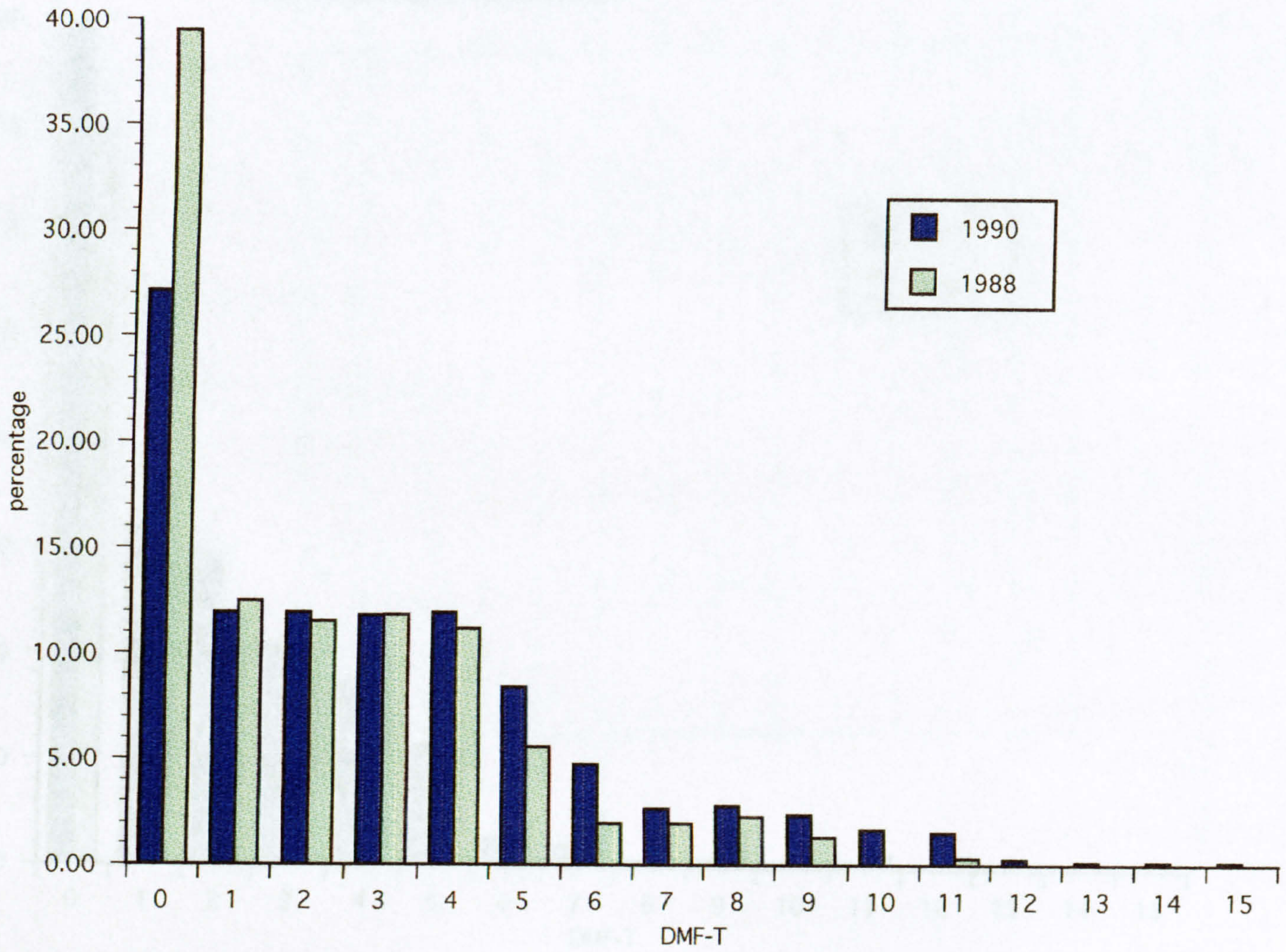
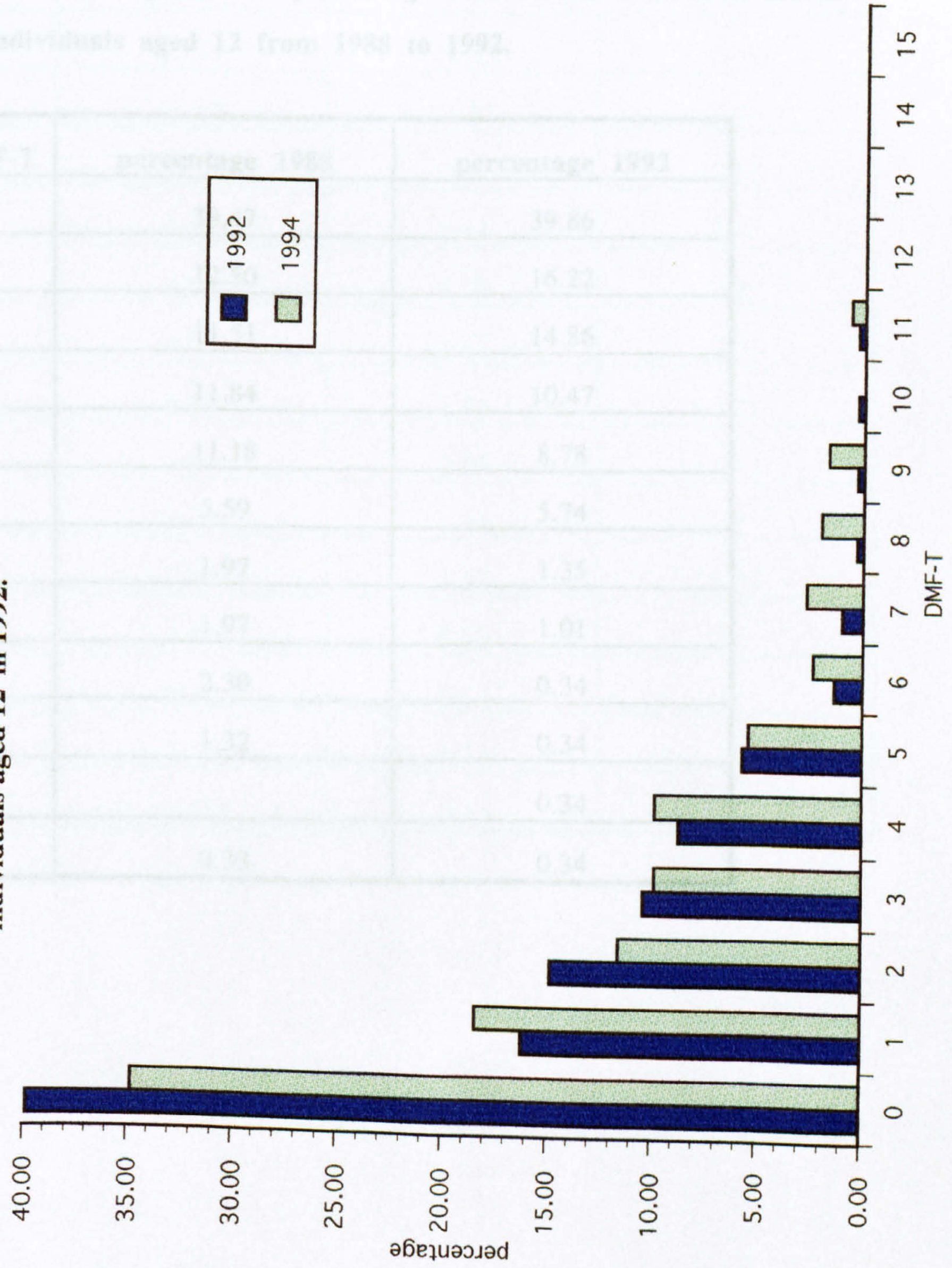


Figure 62: Two year change in the percentage distribution of DMF-T scores for individuals aged 12 in 1992.



the right over the two year period. There is a decline in the percentage caries free, and an elongation of the right-hand tail. These findings are common to the NPDDP analyses: the increase in caries is not confined to an 'at risk' sub group.

Table 25: Changes in the percentage distribution of DMF-T scores for individuals aged 12 from 1988 to 1992.

DMF-T	percentage 1988	percentage 1992
0	39.47	39.86
1	12.50	16.22
2	11.51	14.86
3	11.84	10.47
4	11.18	8.78
5	5.59	5.74
6	1.97	1.35
7	1.97	1.01
8	2.30	0.34
9	1.32	0.34
10	-	0.34
11	0.33	0.34

Table 26: Changes in the percentage distribution of DMF-T scores for individuals aged 14 from 1986 to 1994.

DMF-T	percentage 1986	percentage 1990	percentage 1994
0	14.86	27.14	34.81
1	12.05	11.95	18.43
2	10.44	11.95	11.60
3	14.06	11.81	9.90
4	14.06	11.95	9.90
5	9.24	8.44	5.46
6	8.03	4.78	2.39
7	4.02	2.67	2.73
8	3.61	2.81	2.05
9	4.02	2.39	1.71
10	1.20	1.69	-
11	2.01	1.55	0.68
12	0.80	0.28	-
13	0.40	0.14	-
14	0.80	0.14	-
15	0.40	0.14	-

Table 27: Changes in the percentage distribution of DMF-T scores for individuals aged 12 in 1988 over a two year period.

DMF-T	1988	1990
0	39.47	27.14
1	12.50	11.95
2	11.51	11.95
3	11.84	11.81
4	11.18	11.95
5	5.59	8.44
6	1.97	4.78
7	1.97	2.67
8	2.30	2.81
9	1.32	2.39
10	-	1.69
11	0.33	1.55
12		0.28
13	-	0.14
14	-	0.14
15	-	0.14

Table 28: Changes in the percentage distribution of DMF-T scores for individuals aged 12 in 1992 over a two year period.

DMF-T	1992	1994
0	39.86	34.81
1	16.22	18.43
2	14.86	11.60
3	10.47	9.90
4	8.78	9.90
5	5.74	5.46
6	1.35	2.39
7	1.01	2.73
8	0.34	2.05
9	0.34	1.71
10	0.34	-
11	0.34	0.68

5.5: The hierarchical nature of the caries attack

The analyses of the NPDDP data set suggested that the caries attack was hierarchical in nature: certain teeth and sites were more susceptible than others. In particular, the first molar tooth and the pit and fissured surfaces of the tooth were likely to succumb to a caries attack prior to any other surface. In addition, the order of tooth and site susceptibilities were unaffected by fluoride, sex or age.

This section reports the results of the analyses from the Welsh study. Due to the methodology employed, and in particular the age range of the children examined,

a full mouth comparison is not possible: the children studied being only 9 years old at the date of the last examination. In consequence, the majority of the permanent teeth had not yet erupted. Furthermore, the levels of decay in this population were extremely low, a mean DMF-S of 0.28 at 10 years of age. In consequence, these data compare the susceptibilities of the first molar teeth only.

Table 29 shows the results of the probability analyses for the first molar teeth by surface. Again, as with the NPDDP data analyses, the most susceptible surfaces are the occlusal surfaces of all four molars, and the buccal surfaces of the lower molar teeth.

Table 29: Susceptibilities of first molar teeth by surface

Surface	upper right	upper left	lower left	lower right
distal	0.006	0.005	0.004	0.002
mesial	0.004	0.004	0.004	0.003
occlusal	0.020	0.019	0.020	0.018
buccal	0.005	0.003	0.011	0.010
lingual	0.006	0.006	0.004	0.005

Furthermore, the symmetry exhibited in the previous results is also apparent. There is both a left right symmetry for the all occlusal and lower buccal surfaces and a upper:lower symmetry for the occlusal surfaces only. These results reinforce the issue that, given the similarities in propensity for attack, that at least four surfaces would need to have a sealant placed on them to prevent a single lesion developing: the occurrence of a single caries attack will follow a random distribution pattern.

5.6: Summary

In this section the results of the analyses of the data sets highlight that the epidemiological patterns that were suggested in the conceptual model hold. In particular, the two 'at population' level relationships, that between the caries prevalence and mean DMF score and that between mean caries score and the associated variance, hold true despite possible changes in the conditions under which data were collected.

Furthermore the two 'within mouth' relationships, that between the mean DMF score and the distribution at each DMF score, and the hierarchical nature of the caries attack are also common for at least the first molar tooth sites.

In conclusion, there would appear to be certain laws that govern the distribution and development of dental caries. These distributive laws apply, and do not appear to have changed over the time periods studied and hold for all the populations studied.

6. APPLICATION OF THE CARIES MODEL IN THE SELECTION OF PREVENTIVE STRATEGIES

6.1: Introduction

The caries model has highlighted several relationships that exist to describe the epidemiology of dental caries. There are those relating to the distributive properties: caries can be modelled at a population level and in addition, with knowledge of either the prevalence and/or the mean caries severity, the distribution within the population. Caries can also be modelled at an individual level: for a given DMF score the tooth sites affected will be known. There are also the developmental properties: for a given increase in DMF the new distribution and sites affected can be identified. The next stage of development is the application of this information in helping plan preventive strategies: in essence, what is the most effective combination of preventive agents that should be applied in differing situations. In this section a number of differing scenarios have been developed representing current levels of caries in the UK population for 14 year-olds.

The latest BASCD 14 year-old survey found a range in caries prevalence for the samples studied of between 27% and 90%. Over 90% of the samples however lay in the range between 40% and 80% prevalence. In consequence, the scenarios adopted in this section use three prevalence levels, 40%, 50% and 70% with mean DMF-T scores of 1.00, 1.50 and 2.70 respectively, DMF-S scores of 1.15, 2.20 and 4.20. They are termed low, medium and high respectively.

Each scenario reports the distributive changes of the DMF scores under differing preventive scenarios when compared to a base scenario. The base scenario adopts

current caries status. In addition to each base scenario, three modified distributions are reported. For each of the modified distributions, preventive agents have been utilised and assumptions concerning their effect made. In the first, fluoride is assumed to have a 50% effect, the mean DMF is reduced by half. In the second, fluoride is assumed to have a 30% effect, in the third, a 20% effect. Subsequently, the impact of a fissure sealant strategy for each of the scenarios is also reported. The scenarios described above represent a 'whole population' approach.

In addition to the whole population approaches, a further scenario, the 'risk' scenario is developed. This reports the impact of a differing strategic approach to the problem. No distinction is made between the 'directed population' or 'high-risk' approaches. For the 'risk' approach 'cut off' points at varying percentage points are used. These are 5, 10 and 20% of the population. The results of these latter approaches are then compared to the 'whole population' approach.

In order to reduce caries levels the preventive regimes would need to be applied prior to the age of 14. In an ideal situation the measures adopted should be started as the first molar teeth enter the mouth at about 6 years of age.

6.2: The low caries scenario

6.2.1: The population approach

In this scenario the caries prevalence is 40%, i.e. 60% of the population are caries free. The population would have a mean DMF-T score of 1.00, which

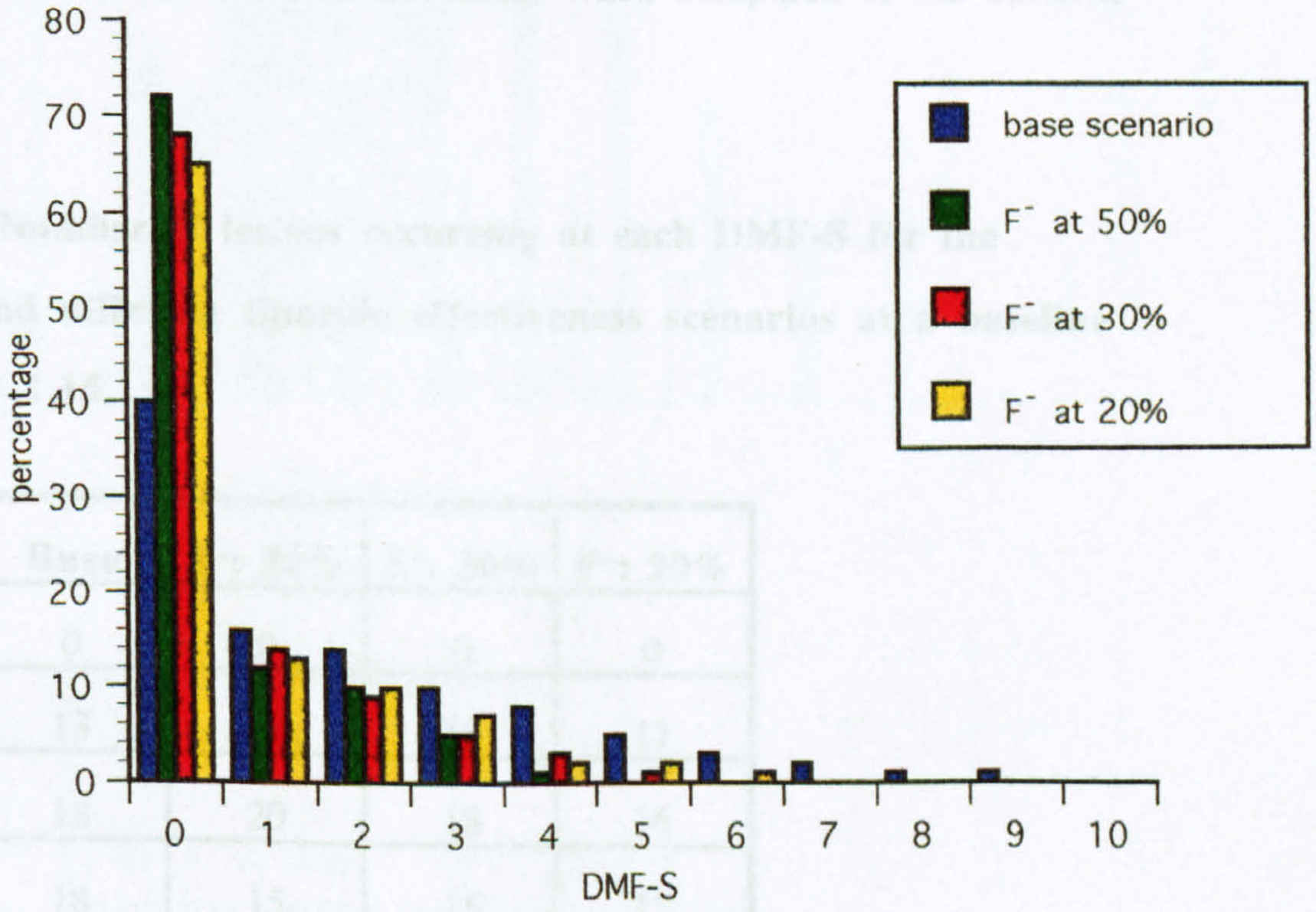
corresponds to a mean DMF-S of 1.15. Table 30 and Figure 63 show the base scenario distribution along with the 3 differing fluoride effectiveness scenarios. The mean DMF-S scores have decreased to 0.57 when the fluoride strategy is assumed to be 50% effective, 0.77 when 30% effective and 0.92 when 20% effective.

Table 30: Frequency distribution of caries for the base and fluoride scenarios for the low caries situation, DMF-S of 1.15.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	60	68	66	65
1	13	16	14	11
2	9	10	9	8
3	6	5	5	5
4	4	1	3	5
5	3		2	3
6	2		1	2
7	2			1
8	1			
9				
10				

There is an increase in the percentage of individuals caries free in all three fluoride scenarios although the overall difference between that in which fluoride is assumed to be 20% effective and that in which it is 50% effective is small; a percentage difference of only 3%. Indeed, at this low level of caries, the percentage changes at any of the DMF-S points is small, although the relative decrease at the higher DMF-S scores is substantial. For example, when compared

Figure 63: Distribution of base scenario and fluoride scenarios for the low caries situation, DMF-S 1.15.



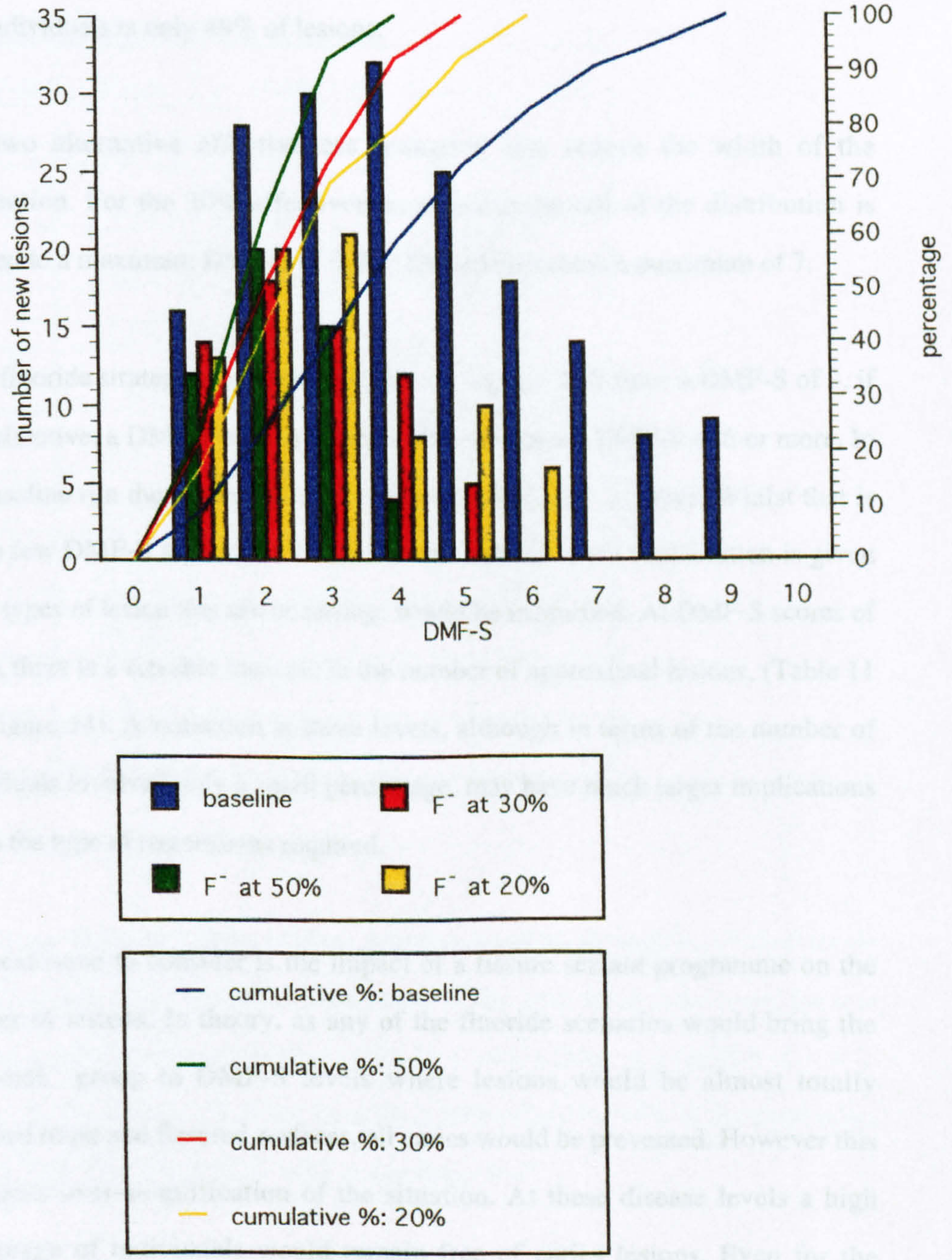
to the baseline scenario, the percentage of individuals at a DMF-S score of 5 or more, the 'high risk' tail, is reduced completely if the fluoride strategy is 50% effective, (Table 30).

Table 31 and Figure 64 show the number of lesions that would occur per 100 individuals under the differing scenarios. For the baseline run the total number of lesions present would be 114. If fluoride is 50% effective in reducing the mean DMF-S score the number of lesions that would occur is 55, a saving of 49 lesions, at 30% effectiveness there is a reduction of 39 lesions, and at 20% effectiveness, 18 lesions would not occur when compared to the baseline scenario.

Table 31: Number of lesions occurring at each DMF-S for the baseline and differing fluoride effectiveness scenarios at a baseline DMF-S of 1.15.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	0	0	0	0
1	13	16	14	11
2	18	20	18	16
3	18	15	15	15
4	16	4	12	20
5	15		10	15
6	12		6	12
7	14			7
8	8			
9				
10				

Figure 64: The number and cumulative percentage of carious lesions for the baseline and fluoride effect scenarios for the low caries situation: DMF-S 1.15



The cumulative percentage distribution of lesions is shown in Table 32. For the 50% effectiveness scenario, the impact of reducing the size of the 'high-risk' tail magnifies the benefits. For example, there is a 100% reduction in the percentage of individuals at a DMF-S of 5 or more but the impact in terms of sites saved per 100 individuals is only 49% of lesions.

The two alternative effectiveness scenarios also reduce the width of the distribution. For the 30% effectiveness scenario the tail of the distribution is reduced to a maximum DMF-S of 6, for 20% effectiveness a maximum of 7.

If the fluoride strategy was 50% effective the highest 19% have a DMF-S of 3, if 30% effective, a DMF-S of 5 or more; if 20% effective a DMF-S of 6 or more. In the baseline run the highest 19% occur at a DMF-S of 7 or more. Whilst that is only a few DMF-S higher, the impact of this reduction, if consideration is given to the types of lesion that are occurring, would be magnified. At DMF-S scores of 7 or 8, there is a sizeable increase in the number of approximal lesions, (Table 11 and Figure 14). A reduction at these levels, although in terms of the number of individuals involved only a small percentage, may have much larger implications due to the type of restorations required.

The next issue to consider is the impact of a fissure sealant programme on the number of lesions. In theory, as any of the fluoride scenarios would bring the 'high-risk' group to DMF-S levels where lesions would be almost totally confined to pit and fissured surfaces, all caries would be prevented. However this is a gross over-simplification of the situation. At these disease levels a high percentage of individuals would remain free of caries lesions. Even for the scenario in which fluoride is only 20% effective, 65% of individuals would not have any carious lesions. The application of sealants to this group would not be a

good use of resources. Furthermore, 11% of individuals would only have one lesion, and 8% two lesions, (Table 31).

Table 32: Cumulative percentage of lesions occurring for the baseline and differing fluoride effectiveness scenarios.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	0	0	0	0
1	11.40	29.01	18.67	11.46
2	27.19	65.45	42.67	28.12
3	42.98	92.73	62.67	43.75
4	57.02	100.00	78.67	55.21
5	70.17		92.00	80.21
6	80.70		100.00	92.71
7	92.98			100.00
8	100.00			
9				
10				

The implications for resources when adopting a sealant strategy in which fluoride was 30 or 50% effective are even greater. Under both these conditions, there is both an increase in the percentage caries free, and in those at a DMF-S of 1 or 2. Indeed, in the scenario in which fluoride is 50% effective, 94% of the population have a DMF-S of less than 3. Why is this important?

The order of tooth susceptibility was highlighted in Section 4.4.2. Due to the similarity in site susceptibilities it is not possible to identify the order in which the

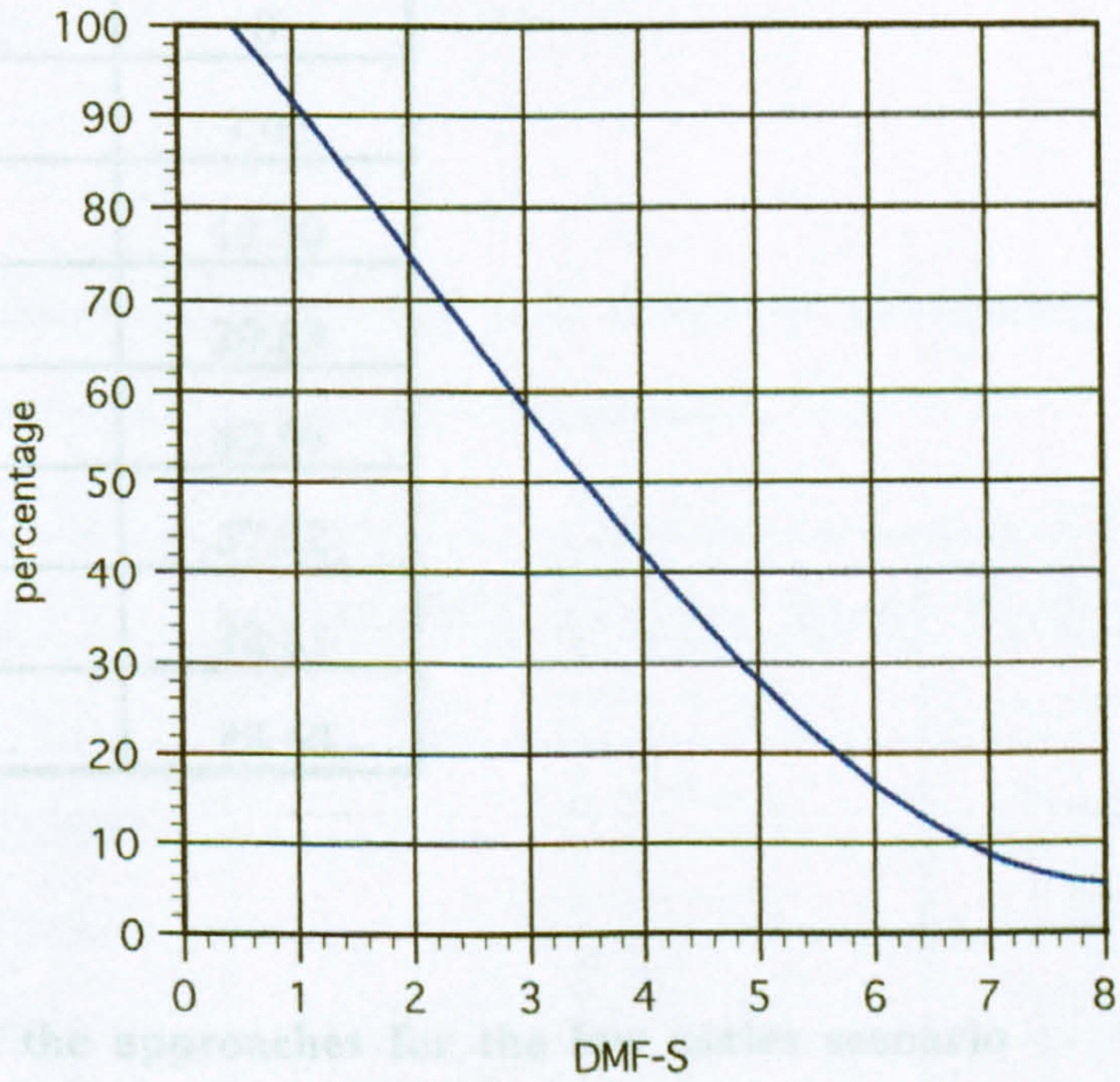
occlusal surfaces of the four first molar teeth or buccal pits of the lower first molars would become carious. The distribution is random. As such, to prevent a single lesion from developing, at least 6 surfaces would have to be sealed. It is only those with six lesions who would obtain maximum benefit from a sealant strategy: in any individual who would not have reached a DMF-S of 6 the strategy is inefficient. The further away from a DMF-S of 6 the greater the inefficiencies. If fluoride was 20% effective only 2% would obtain maximum benefit, whilst at 30% effectiveness, a mere 1%.

6.2.2: The 'risk' approach scenario

Figure 65. shows the relationship between the percentage of sites saved and the definition of the cut-off point of the 'at-risk' group. If the 'risk' group were to be defined as any individual who would be expected to have a DMF-S of 7 or more, just over 7% of sites would be saved. If the 'risk' group were defined at a cut-off point of 5 or more, just less than 30% of lesions would be saved. In all these scenarios two major assumptions are being made. First, there is a satisfactory marker that will allow those who would develop the higher DMF-S scores to be identified, and secondly that any preventive measure was 100% successful.

Obviously as the definition of risk is increased, the percentage of sites saved increases. Table 33 shows the relationship between the definition of the 'risk' group and the percentage of sites saved. If the 'risk' group are those who would have a DMF-S of 7 or more, a total of 7.02% of lesions would have been prevented provided the two caveats mentioned above are accepted. If the 'risk' group is defined as those who would have had a DMF-S of 5 or more, just less than 30% of lesions would be saved, if set at a DMF-S of 4, a saving of nearly 43% of lesions.

Figure 65: The relationship between the definition of the 'risk' group and percentage of sites saved assuming 100% effective intervention for the low caries scenario.



If the 'risk' group were to be defined as anyone who would have a DMF-S of more than 2 nearly 73% of lesions that would have occurred will be prevented.

Table 33: Relationship between the definition of 'risk' group and the percentage of sites saved.

Risk 'cut off' point	% Saving
8	0
7	7.02
6	19.30
5	29.83
4	42.98
3	57.02
2	72.81
1	88.60

6.2.3: Summary of the approaches for the low caries scenario

Table 34 summarises the impact of the various strategies as percentage of surfaces saved and uses 3 percentage 'cut-off' points for any 'risk' strategy: 5, 10 and 20%. At these levels of disease the percentage who would benefit from prevention in any form is limited. Even under the baseline scenario, at 14 years of age 60% of the population would be caries free. The most effective approach is a population approach in which the fluoride is 50% effective and a blanket fissure sealant programme is utilised: the impact of fluoride reducing the DMF-S to 4 even in the most susceptible individuals and these lesions are confined to pit and

fissure surfaces. This however ignores the efficiency limitations of the approach. A high percentage of individuals will gain no benefit, and for many, the benefits may only be small.

Even when only 30% effective the population approach in combination with a sealant strategy will also reduce the number of lesions to zero. With a maximum DMF-S score of 6, the lesions will still tend to be confined to pit and fissured surfaces. It is only when the fluoride strategy is 20% effective will there be a group in which, even with a blanket sealant approach, a small number of carious lesions will occur.

Table 34: Percentage benefiting and effect in preventing caries lesions for the differing strategic approaches

Strategy	% benefiting	lesions saved	% effective
None	0	0	0
F^- 50% and sealants	40	114	100
F^- 30% and sealants	40	114	100
F^- 20% and sealants	40	107	93.86
F^- 50%	40	49	42.98
F^- 30%	40	39	34.21
F^- 20%	40	18	15.79
risk approach: 5%	5	34	29.82
risk approach: 10%	12	49	42.98
risk approach: 20%	18	83	72.81

The 'risk' approach, whilst in theory not suffering from the inefficiencies of the population approach, has practical limitations. At present no screening procedure is 100% effective nor are preventive methods 100% successful. This suggests that, even at best, this approach will have a smaller impact than one in which sealants are used in combination with a fluoride strategy which is 20% effective.

6.3: The medium caries scenario

6.3.1: The population approach

In this scenario the prevalence is 50%: the population has a mean DMF-T score of 1.50, a mean DMF-S 2.20. Table 35 and Figure 66 show the base scenario distribution along with the 3 differing fluoride effectiveness scenarios. The mean DMF-S scores have decreased to 1.10 when the fluoride strategy is assumed to be 50% effective, 1.45 when 30% effective and 1.76 when 20% effective.

With a higher baseline scenario the percentage caries free is lower. For all the fluoride scenarios the impact is more substantial than the low caries scenarios. For example, there is an 11% increase in the percentage caries free between the base scenario and the scenario in which fluoride is 50% effective. In addition the tail is also reduced, although the reduction is marginal for the 20% effectiveness scenario, with 1% of the group having a DMF-S of 11 or more compared to 2% in the baseline scenario, (Table 35).

Figure 66: Distribution of base scenario and fluoride scenarios for the medium caries situation, DMF-S 2.20.

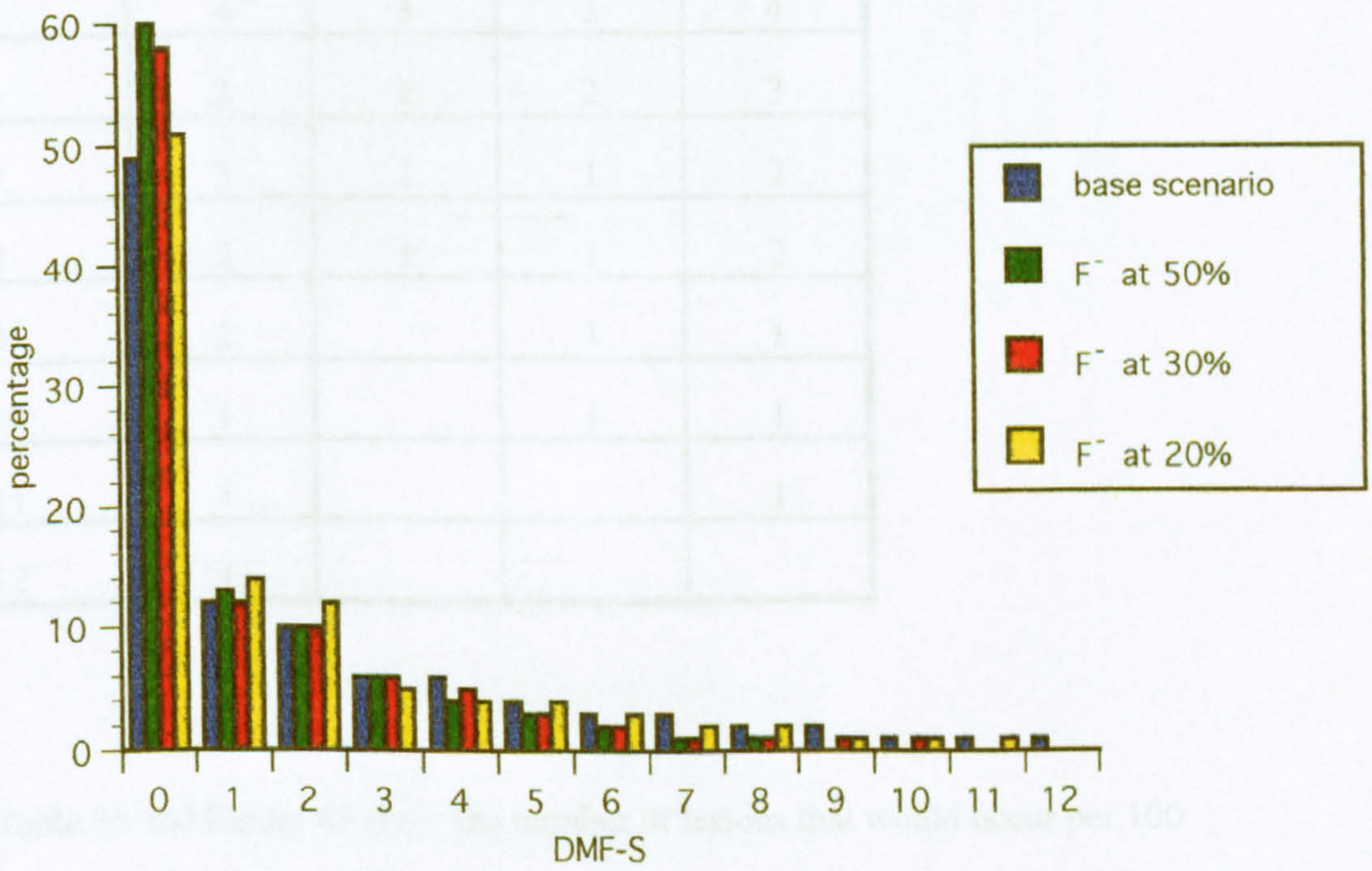


Table 35: Frequency distributions of caries for the baseline and fluoride scenarios for the medium caries situation, DMF-S of 2.20.

DMF-S	Base	F⁻: 50%	F⁻: 30%	F⁻: 20%
0	49	60	58	51
1	12	13	12	14
2	10	10	10	12
3	6	6	6	5
4	6	4	5	4
5	4	3	3	4
6	3	2	2	3
7	3	1	1	2
8	2	1	1	2
9	2		1	1
10	1		1	1
11	1			1
12	1			

Table 36 and Figure 67 show the number of lesions that would occur per 100 individuals under the differing scenarios. For the baseline run the total number of lesions present would be 200.

Under the scenario in which fluoride is assumed to be 50% effective 109 lesions would be found, a saving of 91 lesions. At 30% effectiveness 131 lesions would occur, a reduction of 69, and at 20% effectiveness, 167 would be found, a saving of 33 lesions when compared to the baseline scenario.

Figure 67: Number and cumulative percentage of lesions that occur under the baseline and fluoride effect scenarios in the medium caries situation: DMF-S 2.20.

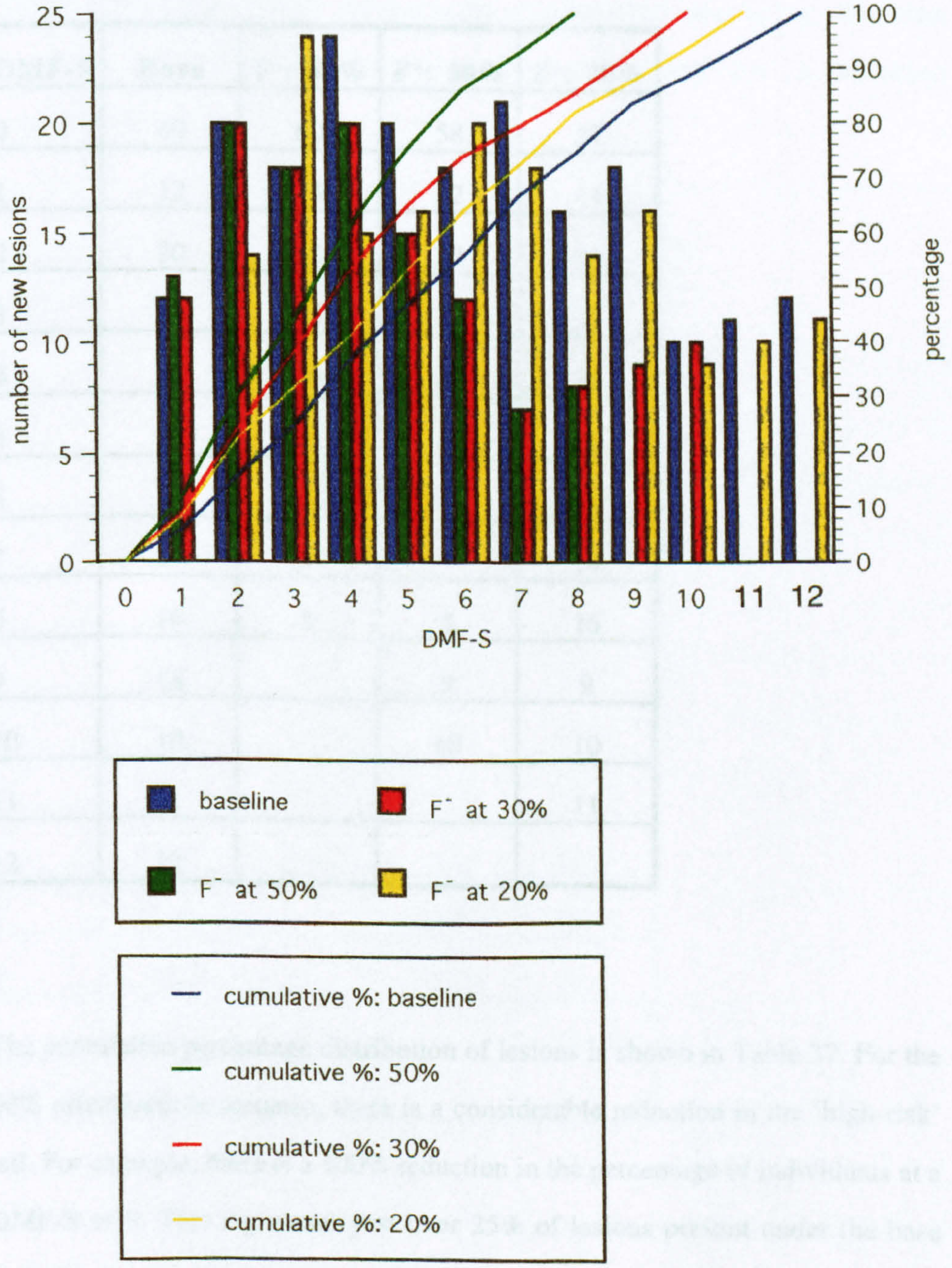


Table 36: Number of lesions occurring at each DMF-S for the baseline and differing fluoride effect scenarios at a baseline DMF-S of 2.20.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	49	60	58	51
1	12	13	12	14
2	20	20	20	24
3	18	18	18	15
4	24	16	20	16
5	20	15	15	20
6	18	12	12	18
7	21	7	7	14
8	16	8	8	16
9	18		9	9
10	10		10	10
11	11			11
12	12			

The cumulative percentage distribution of lesions is shown in Table 37. For the 50% effectiveness scenario, there is a considerable reduction in the 'high-risk' tail. For example, there is a 100% reduction in the percentage of individuals at a DMF-S of 9. This represents just over 25% of lesions present under the base scenario.

As previously, the two alternative effectiveness scenarios also reduce the width of the distribution: for the 30% effectiveness scenario the tail of distribution is reduced to a maximum DMF-S of 10, for 20% effectiveness a maximum of 11.

Table 37: Cumulative percentage of lesions occurring for the baseline and differing fluoride effect scenarios for a baseline scenario DMF-S of 2.20.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	0	0	0	0
1	6.00	11.93	9.16	8.38
2	16.00	30.27	24.43	22.75
3	25.00	44.04	38.17	31.74
4	37.00	61.47	53.43	41.32
5	47.00	75.23	64.88	53.29
6	56.00	86.24	74.05	64.07
7	67.50	92.66	79.39	72.45
8	74.50	100.00	85.50	82.04
9	83.50		92.37	87.42
10	88.50		100.00	93.41
11	94.00			100.00
12	100.00			

If the fluoride strategy was 50% effective approximately fifteen percent have a DMF-S of 6 or more, if 30% effective, a DMF-S of 8 or more, and if 20% effective a DMF-S also of 8 or more. In the baseline run the highest 15% occur at a DMF-S of 9 or more. Again, whilst only a few DMF-S higher, the impact of

this reduction may be considerable. At a DMF-S of greater than 10 there is an increase in the proportion of lesions that occur in smooth and approximal surfaces when compared to, say, a DMF-S of 6.

When compared to the low caries scenario, there is still a group of individuals who will have both approximal and smooth surfaces at one end of the distribution as well as a high percentage of individuals who would remain free of caries lesions. For example, under the baseline scenario, 49% of individuals would not have any lesions and 71% a DMF-S of 2 or less, (Table 35).

Where a sealant strategy would be of most benefit is for those individuals at a DMF-S of between 3 and 8; this sub group first, actually having a number of lesions, and secondly, lesions that are in the majority confined to pit and fissured surfaces. Above this level of caries, there will be the approximal and smooth surface involvement. Whilst the placement of sealants may give some benefit, in that there will be a delay in the development of the pit and fissured surfaces prior to approximal or smooth surface involvement, to provide a satisfactory restoration any sealant is likely to need replacing with a conventional restoration.

For the scenario in which fluoride was assumed to be 50% effective, 60% of the population would obtain no benefit from a sealant strategy as they would be lesion free. Even when fluoride is only 20% effective, over 50% would derive no benefit.

6.3.2: The 'risk' approach scenario

Table 38 shows the relationship between the percentage of sites saved and the definition of the cut-off point of the 'risk' group. If the 'risk' group were to be defined as any individual who would be expected to have a DMF-S of 8 or more,

just over 25.5% of sites would be saved. If the 'risk' group were defined at a cut-off point of 5 or more, just over 53% of lesions would be saved. As with the previous 'risk' scenario the two major assumptions concerning identification and effectiveness are being made.

Table 38: Relationship between the definition of 'risk' group and the percentage of sites saved in the medium caries scenario at a baseline DMF-S of 2.20.

Risk 'cut off' point	% Saving
12	0
11	6.00
10	11.50
9	16.50
8	25.50
7	32.50
6	44.00
5	53.00
4	63.00
3	75.00
2	84.00
1	94.00

As the definition of the 'risk' group moves closer to being defined as 'having caries' the percentage of lesions saved increases. A drop of 2 DMF-S from 10 to 8 as the definition of 'risk' would save an additional 14% of lesions, whilst by reducing it further to 6 would save 44% in total.

If the 'risk' group were to be defined as anyone who would have a DMF-S of more than 2 nearly 84% of lesions that would have occurred will be prevented.

6.3.3: Summary of the approaches for the medium caries scenario

Table 39 summarises the impact of the various strategies as percentage of surfaces saved. Again the percentage of individuals caries free at these disease levels is high, and in consequence, the percentage who would benefit from a more organised preventive strategy is limited. The most effective approach is a population approach in which the fluoride is 50% effective and a blanket fissure sealant programme is utilised: however just over 2% of individuals would develop caries and would be responsible for 8% of the total number of lesions.

A fluoride strategy which is 50% effective would have a similar impact as a 'risk' approach targeted at the 10% at greatest risk whilst one with 30% effectiveness would have a greater impact than a 'risk' strategy aimed at the top 5%.

A 'risk' approach targeted at the top 20% would, at best, only prevent 54% of lesions.

Table 39: Percentage benefiting and effectiveness in preventing caries lesions for the differing strategic approaches for baseline scenario with a DMF-S of 2.20.

Strategy	% benefiting	lesions saved	% effective
None	0	0	0
F^- 50% and sealants	51	185	92.50
F^- 30% and sealants	51	166	83.00
F^- 20% and sealants	51	140	70.00
F^- 50%	51	91	45.00
F^- 30%	51	69	34.50
F^- 20%	51	33	16.50
risk approach: 5%	5	51	25.50
risk approach: 10%	10	88	44.00
risk approach: 20%	20	108	54.00

6.4: The high caries scenario

6.4.1: The population approach

In this scenario the prevalence is 70%: the population has a mean DMF-T score of 2.70, and a mean DMF-S of 4.20. The distributive properties of the baseline and differing fluoride effectiveness regimes are shown in Figure 68 and Table 40. For the scenario in which fluoride was 50% effective, the mean DMF-S has dropped to 2.10; for that in which it is 30% effective, 2.81; and 3.36 in the scenario in which fluoride is 20% effective.

Figure 68: Distribution of base scenario and fluoride scenarios for the high caries situation, DMF-S 4.20

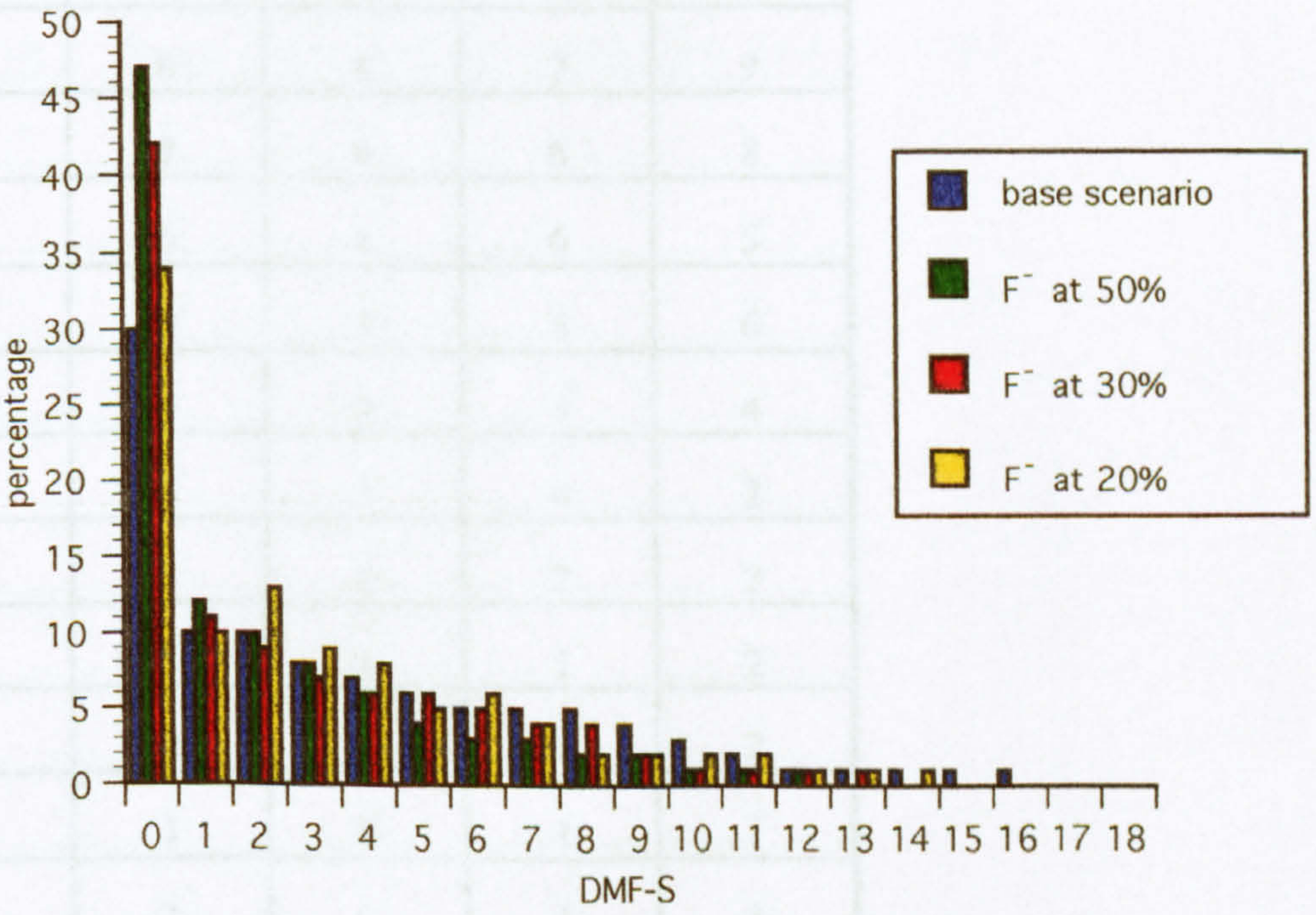


Table 40: Frequency distributions of caries for the baseline and fluoride scenarios, DMF-S of 4.20.

DMF-S	Base	F⁻: 50%	F⁻: 30%	F⁻: 20%
0	30	47	42	34
1	10	12	11	10
2	10	10	9	13
4	8	8	7	9
5	7	6	6	8
6	6	4	6	5
7	5	3	5	6
8	5	3	4	4
9	5	2	4	2
10	4	2	2	2
11	3	1	1	2
12	2	1	1	2
13	1	1	1	1
14	1		1	1
15	1			1
16	1			
17	1			
18				

At these levels of disease even for the strategy in which fluoride is assumed to be 50% effective, the majority of individuals have at least one lesion. However, the

impact of the 50% effectiveness scenario does increase the percentage caries free considerably, from 30% in the base scenario to 47%. As with all the previous scenarios the tail is reduced, and although only by 4% in the best case, given the level of DMF-S is 14 or above, the implications are considerable.

Table 41: Number of lesions occurring at each DMF-S for the baseline and differing fluoride effect scenarios at a baseline DMF-S of 4.20.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	0	0	0	0
1	10	12	11	10
2	20	20	18	26
4	32	32	28	36
5	35	30	30	40
6	36	24	36	30
7	35	21	35	42
8	40	24	32	32
9	45	18	36	18
10	40	20	20	20
11	33	11	11	22
12	24	12	12	24
13	13	13	13	13
14	14		14	14
15	15			15
16	16			
17	17			
18				

Figure 69 and Table 41 show the changing number of lesions and their distribution. In the base scenario there are 425 lesions; 237 in the scenario in which fluoride is assumed to be 50%, a saving of 188 lesions; 296 lesions at 30% effectiveness, a reduction of 129 lesions; and 342 lesions when fluoride is 20% effective.

The cumulative percentage of the number of lesions is shown in Table 42. As in all previous scenarios as the effectiveness of the regime increases there is a decrease in the variance.

At baseline disease levels there is a reduction from a maximum DMF-S of 17 to 13 assuming 50% effectiveness; 14 assuming 30% effectiveness; and 15 at 20% effectiveness. In addition, despite the reduction in disease levels when compared to the baseline strategy, there is a sizeable percentage at a DMF-S greater than 6. Even when fluoride is assumed to be 50% effective more than 50% of lesions would occur in this group of individuals.

Figure 69: The number and cumulative percentage of carious lesions that occur under the baseline and fluoride effect scenarios in the high caries situation: DMF-S 4..20

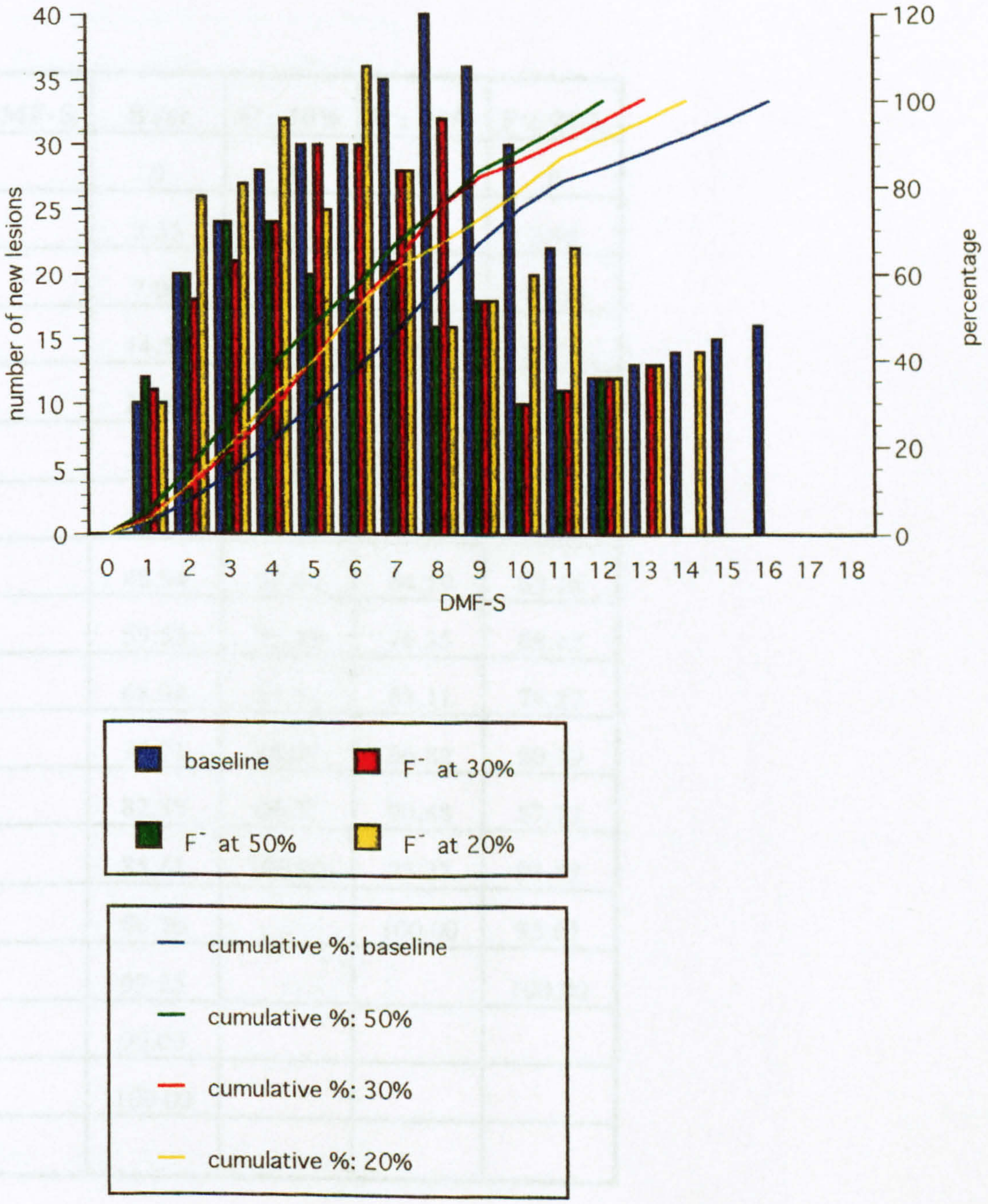


Table 42: Cumulative percentage of lesions occurring for the baseline and differing fluoride effect scenarios for a baseline scenario DMF-S of 4.20.

DMF-S	Base	F^-: 50%	F^-: 30%	F^-: 20%
0	0	0	0	0
1	2.35	5.27	3.72	2.92
2	7.06	14.10	9.80	10.53
4	14.59	28.19	19.26	21.05
5	22.82	41.41	29.39	32.75
6	31.29	47.58	41.55	41.52
7	39.53	56.83	53.38	53.80
8	48.94	67.40	64.19	63.16
9	59.53	75.33	76.35	68.42
10	68.94	84.14	83.11	74.27
11	76.71	88.99	86.82	80.70
12	82.35	94.27	90.88	87.72
13	85.41	100.00	95.27	91.52
14	88.70		100.00	95.61
15	92.23			100.00
16	96.00			
17	100.00			
18				

Table 43: Relationship between the definition of 'risk' group and the percentage of sites saved in the high caries scenario at a baseline DMF-S of 4.20.

Risk 'cut off' point	% Saving
17	0
16	4.00
15	7.70
14	11.30
13	14.59
12	17.65
11	23.29
10	31.06
9	40.47
8	51.06
7	60.47
6	68.71
5	76.18
4	85.41
3	92.94

6.4.2: The 'risk' approach scenario

Table 44 shows the relationship between the percentage of sites saved and the definition of the cut-off point of the 'risk' group for a baseline disease level of DMF-S of 4.50. If the 'risk' group were to be defined as any individual who

would be expected and the measures adopted 100% successful, to limit the highest DMF-S to 15, just over 7.7% of sites would be saved. If the 'risk' group were defined at a cut-off point of 10 or more, just over 31% of lesions would be saved.

The major saving at this level of disease occurs between a DMF-S of 8 and 9. If the cut off point was defined at a DMF-S of 9, 40.47% of lesions would be saved whilst if it was at a DMF-S of 8, 51.06%, a difference of 10.59%.

If the 'risk' group were to be defined as anyone who would have a DMF-S of more than 2 nearly 93% of lesions that would have occurred would be prevented.

6.4.3: Summary of the approaches for the high caries scenario

Table 45 summarises the impact of the various strategies as percentage of surfaces saved. Although the percentage of individuals who would be caries free at these disease levels has decreased when compared to the other lower mean caries levels, the percentage who would benefit from a more organised preventive strategy still remains limited. The most effective approach is a population approach in which the fluoride is 50% effective and a blanket fissure sealant programme is utilised: this would prevent just under 70% of lesions. A similar approach, but one which fluoride is assumed to be only 30% effective provides a 53% saving of lesions.

A fluoride strategy which is 30% effective would have a similar impact as a 'risk' approach targeted at the 10% at greatest risk whilst one with 20% effectiveness would have a greater impact than a 'risk' strategy aimed at the top 5%.

Table 44: Percentage benefiting and effect in preventing caries lesions for the differing strategic approaches for baseline scenario with a DMF-S of 4.20.

Strategy	% benefiting	lesions saved	% effective
None	0	0	0
F^- 50% and sealants	70	296	69.65
F^- 30% and sealants	70	227	53.41
F^- 20% and sealants	70	181	42.58
F^- 50%	70	188	44.23
F^- 30%	70	129	30.35
F^- 20%	70	83	19.53
risk approach: 5%	5	75	17.65
risk approach: 10%	10	132	31.06
risk approach: 20%	19	217	51.06

6.5: Overview of scenarios

The scenarios have highlighted how, even relatively small differences in mean DMF-S scores can have a sizeable impact on the benefits from any preventive strategy, particularly for the 'risk' approach. Tables 45 and 46 and Figure 70 and Figure 71 shows the percentage effect in terms of lesions prevented for the population fluoride scenarios and risk approach with respect to the differing base scenarios.

For the fluoride scenarios there is a reduction in effectiveness in line with effectiveness of the fluoride scenarios: irrespective of the baseline scenario disease level. However, as the baseline disease level falls the impact at 20%

Figure 70: Effectiveness of lesion reduction for fluoride strategies of 20, 30 and 50% effectiveness when compared to base scenarios.

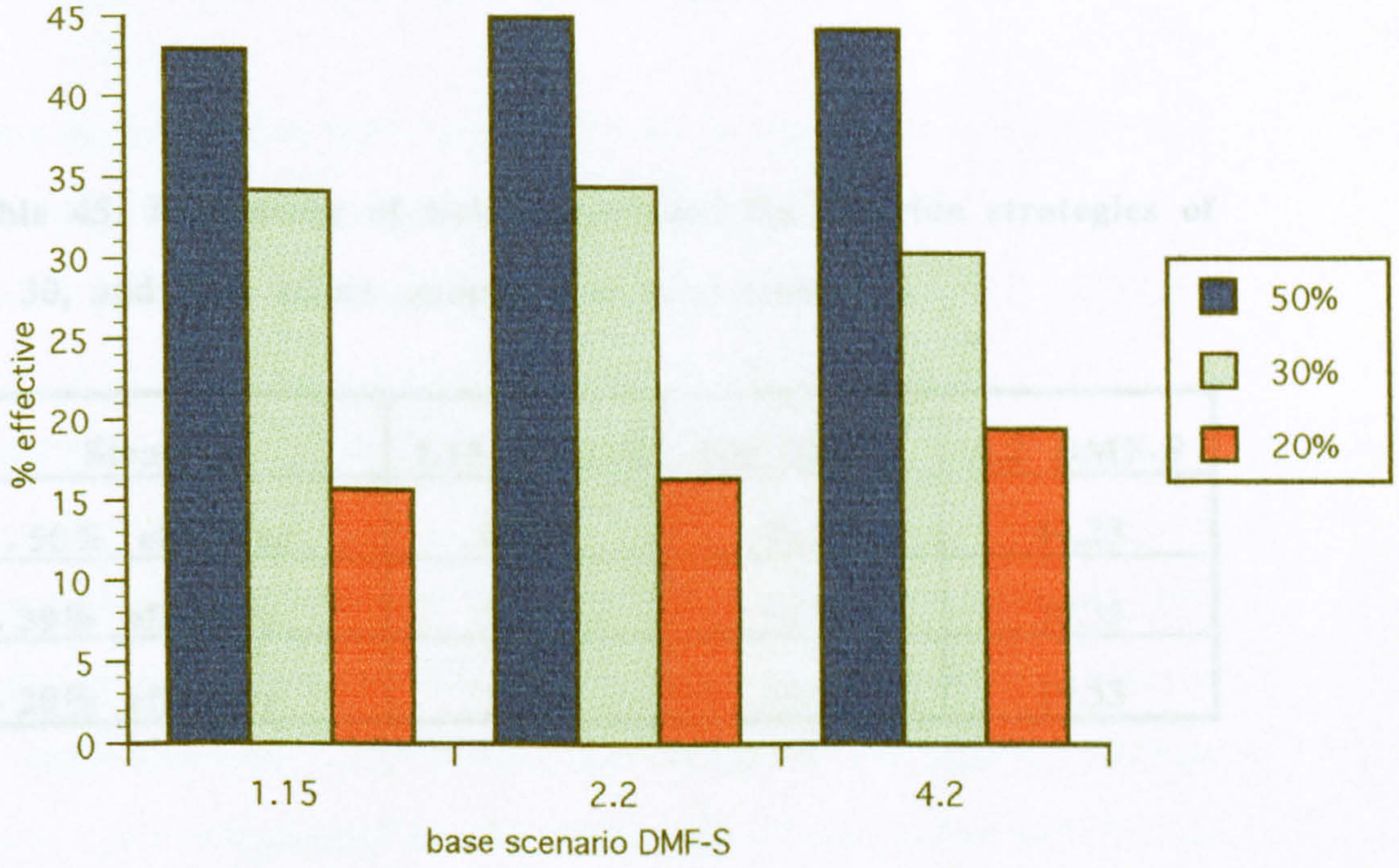
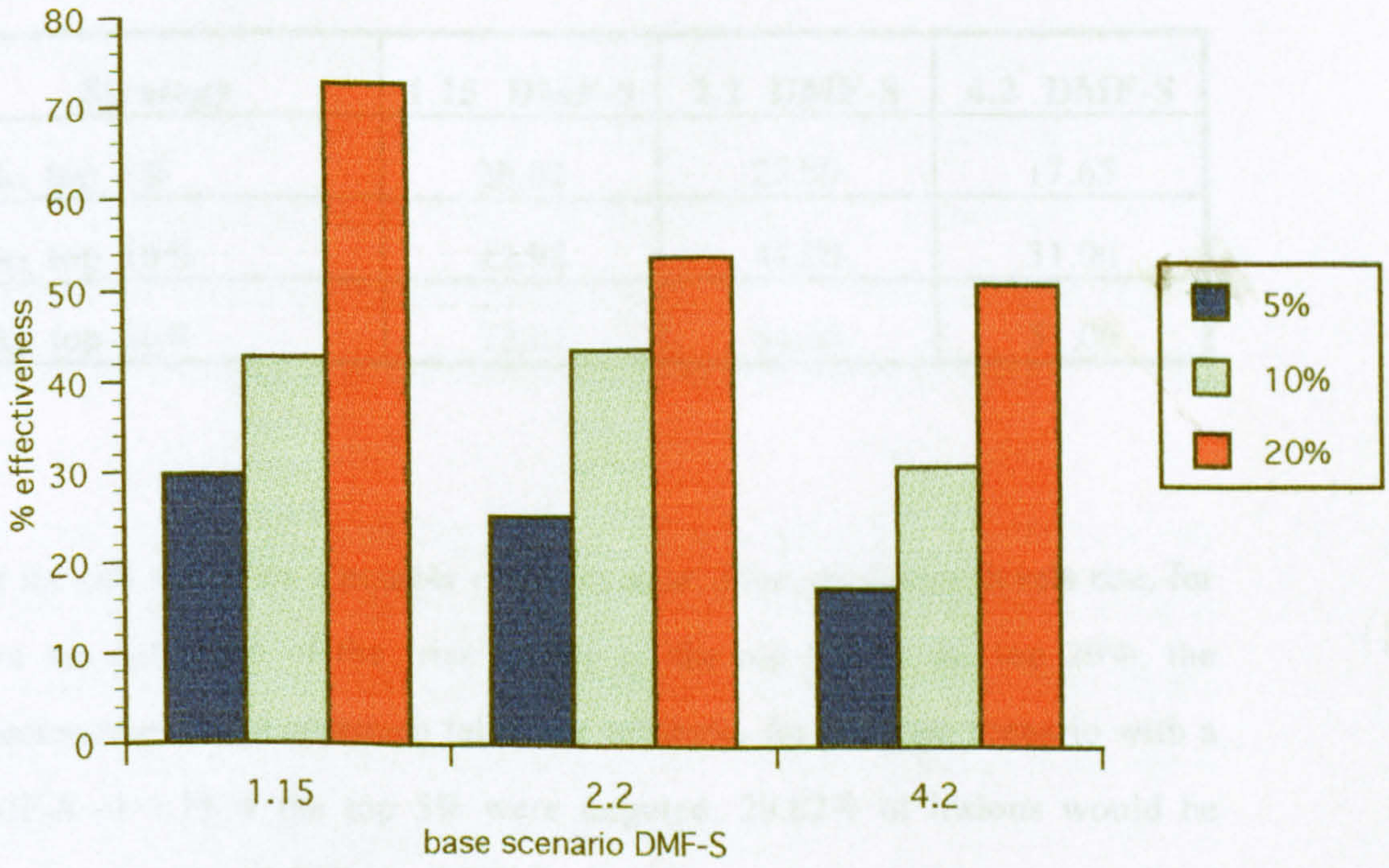


Figure 71: Effectiveness of lesion prevention for risk strategies of 5, 10 and 20% cut-off points when compared to base scenarios.



effectiveness begins to show. Whilst at the high disease level, there is nearly a 20% effect, at the low disease level, this has dropped to just below 16%.

Table 45: Percentage of lesion prevented for fluoride strategies of 20, 30, and 50% effect compared to base scenarios.

Strategy	1.15 DMF-S	2.2 DMF-S	4.2 DMF-S
F^-, 50% effective	42.98	45.00	44.23
F^-, 30% effective	34.21	34.50	30.35
F^-, 20% effective	15.79	16.50	19.53

Table 46: Percentage effect of lesions prevented for risk strategies using 5, 10 and 20% identification points for the differing base scenarios.

Strategy	1.15 DMF-S	2.2 DMF-S	4.2 DMF-S
risk: top 5%	29.82	25.50	17.65
risk: top 10%	42.98	44.00	31.06
risk: top 20%	72.81	54.00	51.06

For the risk scenarios a number of issues arise. First, as disease levels rise, for both the definition of the 'risk' group as the top 5% or the top 20%, the effectiveness of the approach falls. For example, for the base scenario with a DMF-S of 1.15 if the top 5% were targeted, 29.82% of lesions would be prevented against 17.75% at a DMF-S of 4.2. Even if the risk group were defined

by the top 20%, at the high DMF-S the effectiveness is only just over 50%, this improving to 73% at the low DMF-S.

When the definition of risk is the top 10% the greatest impact is at a DMF-S of 2.2, Either side of this disease level the effectiveness drops.

6.6: Summary

This section has shown how the effectiveness of any approach will vary according to the disease prevalence. The scenarios adopted cover the majority of the caries situations found in the United Kingdom. The differing strategic approaches have highlighted how the disease level of the population will effect the outcome. In particular, the variation in effectiveness of the 'risk' approach should be noted.

The population approaches have made a number of assumptions, both in terms of the effectiveness of fluoride and fissure sealants. The lower disease scenarios highlight how, in theory, all lesions can be prevented. However this ignores certain key issues and in particular whether the allocation of resources in this approach can be justified. Given that the majority of the population receive little or no benefit from the preventive measure can the expenditure be justified?

The 'risk' approach also suffers from certain assumptions being made, namely that those 'at risk' can be identified successfully and that the prevention is successful. The previous sections highlighted how those at 'low' risk made a major contribution towards the total number of lesions: the difficulty is not one of identifying a certain percentage but a particular DMF-S.

7: DISCUSSION, CONCLUSIONS AND IMPLICATIONS

7.1: Introduction

This study has highlighted a number of features concerning the epidemiology of dental caries that are relevant in the identification of appropriate, scientifically based caries preventive strategies. First, with knowledge of the mean DMF score the prevalence of dental caries in children will be known, and vice versa. Second, knowing the mean DMF score, the variance can be estimated. Third, for each DMF score, both the teeth and the sites on the teeth affected by caries will be known.

In addition to the epidemiological features of caries the study has also highlighted two other aspects of importance in planning a preventive strategy. First, the effect of fluoride as a preventive agent. The action of fluoride is not site specific: it appears to act by reducing the overall attack intensity. Given that all sites do not have the same vulnerability to caries attack, those with the least, that is the most resistant to decay, will benefit first from fluoride. Second, the benefits obtained by fissure sealant usage depends upon the overall levels of caries. The distributive features of caries imply that any changes that occur are on a population basis: there is not a sub-section of the population who remain unaffected by a change. As the mean DMF score increases the prevalence increases within the population. At high mean DMF scores the majority of the population will have experienced at least one carious attack. With the increase in mean DMF score, the variance also increases. Furthermore, the sites on which fissure sealants act are, in the main, those with the greatest vulnerability to attack. However, due to the relationship between the mean and the variance, the percentage of the population who will benefit from sealants is highly dependant upon the overall caries level. At low

levels of disease a large percentage of individuals will remain caries free and, in consequence, receive no benefits, whilst at high levels of caries, the benefits in preventing pit and fissure lesions may be negated by the development of lesions on the approximal or smooth surfaces of the same tooth.

In this section these findings are discussed. In addition, the methodological issues that may have influenced the findings are discussed and, subsequently, the implications of the findings are compared to the current guidance given by various official bodies for the prevention of dental caries.

7.2: The methodology

7.2.1: Data sources

Three data sources were used in the project: the National Preventive Dentistry Programme (NPDDP) data set, data from studies co-ordinated through the British Association for the study of Community Dentistry, (BASCD), and that obtained through a research project involving the University of Wales School of Medicine and Dentistry and Walsall Health Authority. All of these sources have shortcomings when trying to ascertain the natural history of dental caries as there was some degree of dental intervention in all of them. The DMF index measures both the true caries experience as well as being possible inflated by treatment intervention. Anderson (1989) highlighted this in a study of 12 year-old children in Shropshire after a 25 year interval. The distribution of dental caries, particularly from the earlier study (Anderson et al., 1981), would appear not to be normal, but had a number of modal points suggesting unnecessary dental restorations.

The results from the present study have shown that a number of sites have similar propensities to attack and the practitioners may well have adopted, what is now known as an outdated treatment philosophy. Although the distribution of the NPDDP data exhibits certain similar traits to those found by Anderson, (1989), suggesting that the reported caries experience may be inflated, the level of intervention is very low. Furthermore, although both the M and the F components of the DMF could inflate the natural level, and in particular the former, the disease levels are very low.

These issues are even more pronounced in the Welsh data, with a mean DMF-S score of 0.32 for children aged 10. Although it is not possible to distinguish between the various components of the DMF index from these data, the distribution, with only a single modal value at 0, suggests that any treatment effects are probably minimal.

The statistical methodology adopted, whilst overcoming shortfalls existing in other authors' work, cannot overcome all the issues relating to the differing eruption times of the teeth and the possibility of a phases of enamel maturation (Kotsanos and Darling, 1991). Both these issues may have influenced the outcomes although as the subsequent analyses have shown, the hierarchy of attack is not dependant upon age. This would suggest that under the circumstances studied, the findings are valid. Two circumstances are of paramount importance: low disease levels, inferring a low level of intensity of attack or a high resistance, and the distribution of the transformed proportions responding allowing the adoption of probit analysis. In Section 3, the use of probit analysis was discussed. For this particular procedure to be valid, the data when transformed need to show linearity. Despite the possible problems that dental intervention may have caused, the correlation coefficients obtained

demonstrated a very high degree of linearity, again suggesting that the impact of dental intervention on the findings of this study were marginal.

7.3: The epidemiological characteristics of dental caries

The review of the literature on the patterns of caries highlighted a number of points. First, there was an apparent order in the susceptibility of the tooth sites within the mouth. The findings of this project include the confirmation of the existence of a hierarchy: this is in agreement with much of the work reported. In particular the findings verify the adoption of half mouth methodologies for dental examinations as used in the national survey of the Netherlands (Truin et al., 1991).

Where there are differences between the findings of this study and others, with one notable exception, is the idea of a number of sites having similar vulnerabilities to attack. Section 4.4 highlighted that the susceptibility of tooth sites is not only similar for homologous pairs but grouped sites. For example, at a DMF score of 1, six sites have very similar probabilities of being carious, the pit and fissured surfaces of the first molar teeth. Most authors, whilst accepting that a degree of left:right symmetry exists in caries attack, have assumed that it is either the same site affected on the left hand side of the mouth as on the right (Berman, 1970; DeJong and Dunning, 1971; Wood, 1985; Razak and Razak, 1988; Hujoel et al., 1994). This study found that groups of sites have similar propensities. So, for example in the anterior incisor region, where several sites have similar propensities, the left:right symmetry may have been lost as the mirror image site has not undergone cavitation, but, due to the similarities in susceptibilities, another site on the opposite or even the same side has cavitated. In both cases symmetry will be lost. This finding agrees with the suggestions of Berman

(1970), DeJong and Dunning, (1971), Wood(1985), Rizak and Razak, (1988), and Houjoel et al. (1994) that symmetry exists but not necessarily of homologous pairs. What exists are groupings of susceptibility. This finding is in partial agreement with other authors (Grainger, 1967; Hadjimarkos, 1967; Viegas, 1969; Poulsen and Horowitz, 1974), the difference being that it is not zones of susceptibility but groupings. The posterior teeth do not form a zone but certain sites. This would include certain sites on posterior teeth but in addition may include sites on the anterior teeth.

The idea of groupings has a number of important implications, not least for estimating the cost benefits of a preventive strategy. If the application of a caries preventive strategy lead to a reduction in either the attack intensity or an increase the resistance of the sites to a value at which a particular site was protected, all sites in the grouping would also be protected. Depending upon the magnitude of the grouping, several sites may be protected. This may offer an explanation as to apparent ease at which DMF levels fell at the beginning of the decline seen in many industrialised countries since the 1970's. The application of preventive agents allowed all sites in the least susceptible groupings to fail to succumb at the attack intensity. Figure 13 suggests that the least susceptible groupings contained considerable numbers of sites. Thus, a reduction in the attack intensity which benefited this grouping would lead to substantial savings in cavitated sites.

The change, reported by many authors, in the changing ratio of smooth, approximal on the one hand, and pit and fissured surfaces on the other is in agreement with this study's findings (Burt, 1985a; Dummer et al., 1990; Vehkalahti et al., 1991). In Section 4.4, the changing ratios were reported by DMF-S. For example, at a DMF-S of 1, the ratio of pit and fissured to smooth or approximal surfaces is 99:1, at a DMF-S of 10, the ratio had changed to 3:1. However, the changing ratios should be set against the overall decline in caries.

The reduction seen is not similar for all sites: certain sites remain more likely to have experienced caries than others. Most importantly, this study has identified that fluoride does not influence the order of site susceptibility. The data available does not enable a clear distinction to be made between possible topical or systemic effects, although the data suggest that the pattern is independent of the methods of delivery. This suggests that the effect of fluoride is to reduce the overall intensity of attack. All sites benefit rather than different sites benefiting to varying degrees. For example, in Section 4.4, the order of susceptibility for the differing sites was independent of both water fluoridation status and programme site. In certain of the programme locations topical and systemic fluorides were used and the hierarchy of vulnerability remained similar. This contradicts the finding by Ripa et al. (1985) and others, (Backer Dirks et al., 1961b; Horowitz et al., 1972), but agrees with McDonald and Sheiham (1992). Ripa (1985) and others (Backer Dirks et al., 1961b; Horowitz et al., 1972), however, appear to have assumed that all sites have a similar initial susceptibility.

It has not been possible to fully answer the question concerning the existence of a period of maturation for enamel and whether variation exists for a site as enamel matures. However, the impact of age, as with fluoride, seems not to influence the order of susceptibility. Given that sites have different susceptibilities, even if variation did exist, unless the changes in variation were sufficiently large enough to alter the inherent differences in site susceptibility, no changes in the hierarchical pattern would be seen.

Overall the findings confirm that there is a hierarchy by tooth type and site in the pattern of dental caries attack. While it is not possible to identify precisely the order that each tooth or site will succumb to a caries attack, groups of sites or teeth can be placed in a hierarchy of risk. This develops further the arguments proposed by Viegas (Viegas, 1969), Grainger (Grainger, 1967) and Poulsen et

al. (Poulsen and Horowitz, 1974) of a hierarchy of 'within mouth' zones. In addition neither the level of fluoride or the age of the individual alters this developmental pattern to any major extent.

7.4: Preventive strategies for caries

7.4.1: Strategy approaches

Rose (1985) when examining the basis for the aetiology of disease identified two distinct issues; the determinants of individual cases, and the determinants of incidence rates. He went on to argue that there were two distinct strategies, the high risk approach, which seeks to protect susceptible individuals and the population approach, which seeks to control the causes of the incidence. These arguments can be refined for the prevention of dental caries into the population approach, which will adopt measures for everyone and the risk approach, in which either groups of individuals can be targeted, the directed population strategy (Sheiham and Joffe, 1992), or the high risk approach, in which individuals are targeted. The question for the policy maker is what combination of these strategies should be adopted and the emphasis given to each of these strategies within an overall approach. For maternal and child health, the WHO have adopted the 'risk approach' (World Health Organisation, 1978). In this approach it is stated that:

'Individuals and groups with an increased expectation of complications or disease are defined as being "at risk" and the aim of the health services should be to identify them as early as possible and to intervene in order to reduce the risk'

The logic behind this approach requires a number of requirements, namely a mechanism to identify individuals or groups 'at risk' that conforms to the WHO guidelines on screening (World Health Organisation, 1971), and, as highlighted in this thesis when discussing economics, that the costs of such an approach can be justified. First, the approach will be discussed.

Hayes (1991) challenged the adoption of the 'risk approach' and highlights the shortcomings from two aspects; the prediction of future outcomes and the ability of strategy users to measure markers. Of particular importance is the former. Hayes raises four specific concerns: the assumed regularity of empirical effects; the status of relationships between risk factors and defined outcomes; the difference between social and natural risk factors, and; the temporality of prediction. This thesis was not concerned with the performance of screening programmes although Hausen et al. (1994) have provided a summary of the current status of caries prediction. In their conclusions, they commented that the whole population approach should still be adopted as services are unable to provide adequate individual protection to those at greatest risk and that dental caries remains a common disease. But perhaps more importantly they highlighted, as Hayes had previously done, that the inadequacies of current screening mechanisms, in particular the shape of the relationship between previous disease experience and that of disease into the future, have not been overcome. Although longitudinal data over a relatively short time frame were used in the present study, the results have highlighted that the relationship between existing disease levels and future caries increment is non-linear, the shape of the curve reinforces the argument that everybody remains at risk.

Although there is a positive relationship between the mean future incremental DMF score and initial DMF score the magnitude of the differences are small. While those individuals at an initial low DMF score had, on average, a lower

caries increment than those individuals with a high DMF score, both groups developed caries. For example, for the NPDDP group which received both fluoridated water and fissure sealants, those initially at a DMF-S score of 0 had an increment of 0.67 compared to an increment of 2.38 for those with an initial DMF-S of 7 or more, (Table 20 and Figure 42). If consideration is given to the distributive properties of caries within the overall population, as highlighted in Section 4.3 on the epidemiology of caries, the impact at a population level of the differences, in terms of the number of lesions at each initial DMF, is less distinct. For the higher percentage of individuals at the lower disease level, the overall number of lesions for each initial disease point are similar. The question for the planner is now whether a utilitarian approach should be adopted, namely that Society would benefit by reducing every individual's risk, or whether, because the lesions found on the changed tail of the distribution will involve more sites, and in consequence are likely to incur greater treatment costs, that an alternative approach should be adopted.

This highlights the importance of selecting the outcome measure on which to assess the justification of strategy selection and makes answering the question 'Which strategic approach?' very difficult. Is it the impact on individuals or on the population at large, or more pertinently, to what extent will economics influence the decision? Indeed, as Fejerskov (1995) suggests the question may be better stated as 'What combination of strategic approaches?'.

The findings in Section 6 help answer this question. The distribution of caries within the population at low levels of caries highlights the considerable number of individuals who would appear not to benefit from caries preventive measures. However, the distributions shown are of 14 year-olds and further increments in caries would be likely. That apart, the appeal of a 'high' risk approach is seductive. Certainly, the future increments that will occur within this population

would suggest that large numbers of individuals will remain caries free into adulthood: preventive measures applied to these individuals would be a waste of resources. Table 34 summarises the impact of the varying approaches and contents. The 'risk' approach aimed at the 10% at greatest risk provides a similar impact in terms of overall effectiveness as a population strategy in which water fluoridation is assumed to be 50% effective. The costs of the two approaches would need to be considered. The costs of screening and the consequent application of preventive agents to the 'risk' group would need to be balanced against the costs of implementing fluoridation.

For the medium caries scenario, a mean DMF-T of 1.50 and mean DMF-S of 2.20, the strategic approach in which fluoride is assumed to be 50% effective has a similar impact as a 'risk' approach aimed at the 10% at greatest risk. Again the economics of the two approaches need to be compared. However, due to the higher mean DMF scores, the distribution has altered. Higher number of lesions, both in absolute and relative terms, indicate that a directed population approach might be feasible. By targeting sealants at the group with between 3 and 8 lesions as opposed to all individuals, the benefits accrued may justify the costs.

At a high level of caries, a mean DMF-T of 2.70 and mean DMF-S of 4.20, the arguments for a 'risk' approach are less clear. Tables 46 and 47 illustrated the issues. The definition of the 'risk' group influences the results considerably. Concentrating on the top 5% at any of the levels studied tackles less than 30% of the problem, and as the mean DMF score increases, the percentage benefiting decreases. For the 'population' approach, considerable variation exists for the differing levels of effect reported. The variation at the differing caries levels, however, is far smaller, than for the 'risk' approaches.

In summary, at low levels of disease, the crucial factor in any justification for adopting a preventive approach would appear to be cost, although the real effect of fluoride would need to be considered. In addition the costs of adopting a treatment strategy should also be considered when making any decision about how to tackle the problem. At medium levels of caries, a 'directed' approach for sealants has validity providing the economic arguments can be justified. The distributive changes seen when compared to that occurring at low DMF levels mean a greater percentage will obtain benefit. At high levels of caries, a population approach is justified. The percentage who receive no benefit decreasing to 30% by 14 years of age.

7.4.2: Strategy content

The strategy content consists of three main components: fluoride, fissure sealants and the control of dietary sugars. Although these items were discussed at length at a recent symposium of caries preventive strategies (Curzon and ten Cate, 1993), the papers presented, with one exception, failed to use the epidemiology of the disease to assist in formulating a strategy. Furthermore, even in the single presentation that did highlight how the pattern of caries had changed, the authors failed to identify anything but a blanket approach for sealants and intensive professional mechanical tooth cleaning for individuals identified as 'high' risk, (Axelsson et al., 1993). No costings for the programme were supplied. The symposium presented the findings of several authors work dividing the sessions according to preventive agent. While a number of authors highlighted the benefits of a population approach, considerable discussion and emphasis was placed on identifying individuals at 'risk'. However, nowhere was the definition of at 'risk' quantified, except to suggest those with the existing highest disease levels. No discussion of existing levels of fluoride, or on how the impact that sealants have

at differing disease levels might vary was mentioned. This study has overcome these deficiencies in allowing the quantification of lesions to be modelled for differing disease levels. For example, the study has highlighted the distribution of a population at differing disease levels which would identify the percentage of individuals who would benefit from sealants.

In a special supplement of the American Dental Journal (American Dental Association, 1995) a risk approach was adopted that involved assessing individuals, using a five stage process to develop the appropriate content. The first three stages identified the strategy content. First, the existing potential for development. This included measurement of the existing DMF-S, tooth morphology, usage of medication that would decrease saliva flow, and any other medical treatment or condition. Second, from the previous step, assigning an individual to a risk category and subsequently identifying what risk factors could be modified. The first of these was establishing whether an individual was exposed to sufficient systemic or topical fluoride. For any individual who had developed a single lesion within the last twelve months, and would subsequently be classified, at minimum, at moderate caries risk, sealants and mouthrinse would be prescribed.

Again this fails to utilise the epidemiological characteristics of the disease. The FDI/WHO approach (Federation Dentaire Internationale, 1984) also suffers from this shortcoming. The guidelines for fluoride usage include supervised mouth rinsing, including gels and varnishes which has been challenged by both Disney et al, (1990) and Petersson (1993). Perhaps an even more serious omission is the lack of what constitutes low or high levels of caries within the document. Indeed, the report seems to suggest that it is the complete eradication of caries that is the goal of any strategy. From the data reported, more recent guidelines have suggested a risk approach (British Society of Paediatric Dentistry, 1993;

Woodward et al., 1995). However these operate at an individual level and make the assumption that individuals at a particular risk will continue to remain at risk. This implies that the group form a distinct tail. As the data from this thesis have shown the distribution of future caries increments suggest that every individual is at risk: there is a population shift as caries levels rise. Indeed as Norbland (1986) concludes 'The possession of a caries-free dentition under the age of 10 is not a good predictor of zero caries incidence thereafter'.

Fissure sealants are widely recommended as integral to the content of a caries preventive strategy. The impact of sealants appears not to be dependant upon the overall attack intensity. The caries increments in both 7 and 11 year-olds across the DMF-S groupings were remarkably similar (Tables 19 and 20). This appears to confirm that, by simply blocking the susceptible sites with a sealant, the overall attack intensity is not reduced. The increments continue to rise in line with previous disease levels: the approximal and smooth sites are attacked (Tables 19 and 20). Thus when considering selection of sealants as part of a strategy, a planner should consider what the existing caries situation is: what will the status be of the first molar pit and fissured surfaces of the current 7 year-olds in 5 years time? This can be answered by looking at current 12 year-olds, and assuming that if nothing is done, the situation will remain constant. By looking at the distribution of disease one can make an assessment as to whether, the number of children benefiting can justify the fissure sealant programme. At low levels of disease, with increments also likely to be low, the number of individuals who remain caries free is likely to be substantial, whilst at the other extreme, if disease levels are high, the development of caries on smooth and approximal surfaces will negate most of the benefits in the reduction of pit and fissure lesions.

Fluoride is the most potent of the preventive agents for caries. This is an unequivocal finding from this study. Indeed, one possible explanation for the

population changes seen in this study is the widespread availability of fluoride. However, the study has highlighted how, simply increasing fluoride exposure, does not necessarily bring a corresponding reduction in disease; the response is not linear. Spuznar and Burt (1990) highlight the problem that fluoride exposure may be increasing overall and there is a limit to the effect of fluoride. Therefore future reductions will be less likely. Bohannan et al. (1985) had also contested the wisdom of simply applying fluoride. This study has not attempted to quantify the dose response relationship but has suggested improvements in oral health that would occur at varying levels of effect. Again, using the knowledge of the epidemiology of caries, planners can now quantify the cost effectiveness of prevention when analysed against a treatment strategy using assumed levels of effectiveness for fluoride. This study has shown that the distribution of carious lesions can be modelled for a given DMF score. Consequently, with knowledge of both the costings of a caries preventive strategy and that of treatment, a valid comparison can be made. The model would start with the present situation for a given age cohort and the distribution of both the existing situation and that in which no changes in strategy were adopted identified for the time period under study. The distribution of new carious lesions would then be quantified. Assumptions can be made about the quality of treatment and the effect of fluoride and impact of sealants. This would allow planners to apply scientific principles to the selection of caries preventive strategies.

7.5: Conclusions

1. The mathematical relationship $y = 42.0 * x^{0.51}$. describes the relationship between the prevalence of dental caries in the permanent dentition and the mean DMF-T score. Furthermore the relationship $y = 1.53* x^{0.55}$ describes the

relationship between the variance and mean DMF score. In consequence, knowledge of either the prevalence or mean DMF score of a given population can be used to provide an accurate estimation of the distribution of DMF scores within that population.

2. The development of dental caries by tooth site and tooth type is hierarchical in nature. The most susceptible tooth sites, the pit and fissured surfaces of the first molars will cavitate first and the least susceptible, the smooth surfaces of the canine teeth, last. The hierarchical pattern is independent of fluoride; fluoride reduces the overall attack intensity but is not site type specific in its action.

3. The above two conclusions when combined allow the distribution of dental caries within a population to be modelled at a 'within mouth' level. The model when used provides information on the changes in both the number and type of sites saved and the percentage of the population at each DMF score. This can be used as a basis to provide a scientific approach for the formulation of caries preventive strategies.

4. Careful thought needs to be given before adopting the usage of fissure sealants as part of a preventive strategy. The reasoning is twofold. First, the distributive properties of caries within the population indicate that, at low levels of caries, only a relatively low percentage of people would benefit, whilst at high disease levels substantial numbers of a given population will develop approximal lesions in those teeth which would be sealed, which may negate the benefits of sealant placement.

5. Current shortfalls in knowledge relating to the effectiveness of differing fluoride regimes prevent their impact being modelled accurately. Research is required to

address this shortfall so that the model can be used to quantify the impact of differing strategies both in terms of sites saved and for cost benefit purposes.

As Rose (1980) comments in response to the question "Which strategy is right?", the answer is that '..each has its distinctive contribution, and each has its costs and limitations'. While no attempt has been made to quantify the costs that Society feel are appropriate, this project has provided a scientific basis for caries preventive strategies. In doing so, it has highlighted the limitations of current recommendations concerning strategic approaches, and as such provides valuable data to assist in the formulation of what remains a major public health problem.

7.6: Implications and suggestions for further research

1. The study has highlighted certain basic 'laws' that govern the distribution of dental caries. Although the collection of data through epidemiological studies using current methodologies provides knowledge of the relative balance of various components of the DMF index, this may not be of relevance under certain circumstances. Where data are required to estimate the overall caries experience alone, this study suggest that examination of the first molar teeth alone would provide adequate. The recorder would identify simply whether a carious attack had occurred. This may provide a more cost effective method of data collection, in particular parts of the World where data are currently lacking for the planning of care. It is suggested that research into the cost and feasibility of obtaining data through a simplified examination process is undertaken.

2. There is a lack of data on the effectiveness of differing combinations of fluoride regimes. This shortfall limits the application of the model produced in this thesis

at present. A systemic review of the effectiveness of fluorides should be undertaken, the results of which should be incorporated into the model to allow planners to base decision making on more scientifically valid scenarios.

3. The study has identified a major shortfall in the application of health economics to caries preventive strategies. The quality of the literature is very poor, possibly due to the lack of information for modelling caries. While the study has overcome the shortfall in modelling, the work on cost-effectiveness of fluoride remains a necessity. In addition, the costs of implementing sealant strategies and providing treatment need identifying.

4. The economic models do not identify all intangible benefits. The impact on the individual of differing disease levels should be measured. As caries levels increase there is a change in the balance of pit and fissured surfaces to approximal and smooth surfaces. This change may also be indicative of an increase in oral problems that the individual suffers; larger restorations requiring more frequent replacement, more pain, and an increase in extractions. Indeed it is possible that at low levels of disease the costs of efforts to reduce caries further outweigh the benefits. Research is required to establish both at a population level and at an individual level how the magnitude of oral health problems alter according to the prevalence of the disease.

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